THE
LARYNGOSCOPE.

VOL. XXXIX DECEMBER, 1929. No. 12

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE TONSILS, AND SOME EXPERIENCES OF THEIR SURGICAL TREATMENT.*

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Mr. President and Gentlemen:

To be invited to take an active part in a Clinical Congress of the American College of Surgeons is a distinction highly esteemed by my British confrères and as your honored guest on this occasion, I ask you to believe how very deep is my appreciation of the compliment you have paid, not only to myself but also to my fellow workers in the Old Country.

To enter your Holy of Holies has for a long time been one of my cherished hopes because I believe that, in its earliest days, Sir Rickman John Godlee, then President of the Royal College of Surgeons of England, presented to your College tokens emblematic of the good will and friendship of the older for the younger brother. He was one of my revered teachers, and later on my senior colleague on the Staff of University College Hospital, London, and in each of these capacities always my guide, philosopher and friend. In some more exalted sphere of existence, I feel sure that he rejoices at the honor which you are conferring today on one of his former and very humble pupils.

Coming now to the subject of discussion, it may seem strange to you that having been given the whole field of oto-rhino-laryngology

*Read before American College of Surgeons, Chicago, Oct. 16, 1929.

Editor's Note: This ms. received in the Laryngoscope Office and accepted for publication Oct. 18, 1929.

from which to make a choice, I should select a subject so commonplace, threadbare, and even risky as anything to do with the tonsils.

I say "risky" because one might visualize many of your Fellows bringing their own self-designed and foolproof guillotine "which enucleates the tonsil complete in its capsule" and, needless to add, the operation would be a bloodless one. Since my arrival in your hospitable country I have heard used the term "tonsil kings" but no definition of this human species having been forthcoming, I have had to draw my own conclusions. If any such potentates be here today, I must ask their forgiveness if the mechanical aspect of tonsil surgery finds little place within the compass of my short address.

AN ANATOMICAL MISNOMER.

The term "supratonsillar fossa" is often used when matters connected with the tonsils are being discussed. Since that potential space is lined with epithelium, contains more or less lymphoid tissue and is within the capsule of the tonsil, it would be more correct if we dropped the Latin prefix and simply spoke of the "tonsillar fossa".

SOME SURGICAL ASPECTS OF DEVELOPMENTAL ANOMALIES.

On occasion some of these may become of surgical importance, and probably there are several of you here who have been hindered during a tonsil operation by an unduly long and ossified stylohyoid ligament. I have not known it to prove a serious complication because by separating and pushing outwards the surrounding gland or connective tissues, the projecting bony spine can easily be divided with forceps and removed.

Less commonly, we may meet with an isolated spicule of bone or cartilage. The large majority of these are, in the opinion of the late Professor S. G. Shattock, embryonic "rests" of the branchial arches.

I would like to take this opportunity of showing you an unique specimen of a bony tumor or "osteoma" which bulged forward the left soft palate and invaded the corresponding tonsil so that only a small portion of this remained in the lower end of its recess.

I saw the patient when she was 5 years of age and it was agreed to wait till she was older before attempting its removal. I did not see her again for 25 years, when she consulted me because the tumor was ulcerating through the soft palate and by scraping the side of the tongue made it sore. A crucial incision over the area of ulcera-

tion and separation of the surrounding soft tissues allowed easy enucleation of the osteoma. It was as large as a walnut and measured 1½ inches in its long diameter.

Its pressure on the cartilaginous portion of the Eustachian had destroyed the median half of that structure and without producing any ear symptoms. So rare was such a specimen that not even Sir John Bland Sutton, with his unrivaled experience of developmental curiosities, had seen anything like it, and the late Mr. Howard Mummery at first glance thought it suggested an odontome. He ground down many sections and reported that it contained no dentine but that sections of its fibrous capsule showed the presence of bone and cartilage deposits.

Professor Shattock then carried out a long and patient research on the nature of the tumor and his full and admirable report on it you will find in the *Journal of Laryngology and Otology*, April, 1924. It was included there by Dr. Irwin Moore in his exhaustive contribution on "Osseous and Cartilaginous Formations in the Tonsil".

It will suffice now to say that the tumor was composed almost entirely of compact bone.

TONSILLAR CALCULI.

Although neither osseous nor cartilaginous in structure, one may refer to these before passing to other matters because attention has just been drawn to the occasional presence of bone and cartilage in tonsils.

Calculi may vary in size from a millet seed to that of a bantam's egg and weighing as much as 1 ounce. Their composition is mainly phosphate and carbonate of lime and magnesia.

They may cause pain in the tonsil on deglutition and tend to produce attacks of tonsillitis, but my reason for mentioning them at all is this. If a small calculus be hidden in the tonsil and give rise, as it often does, to neuralgic pains in the depth of the aural meatus the causation of this symptom may easily be overlooked. Not infrequently I have seen the condition diagnosed as "a gouty" or "rheumatic" throat with consequent ineffective and expensive treatment, especially when this involved a prolonged stay at a fashionable spa.

There should be no particular difficulty in establishing the presence of a calculus in the tonsil because the latter is generally redder than its fellow and pressure on its anterior surface causes pain. A

blunt probe passed into the tonsillar fossa or into some of the upper lacunae will rarely fail to detect the rough surface of the tonsillolith. Small ones can easily be removed by a small hook or spud, the larger and buried ones may have caused such a degree of tonsillar inflammation and sepsis that enucleation of the whole gland will be the best form of treatment.

SURGERY IN ITS RELATIONSHIPS TO THE FUNCTIONS OF THE TONSILS.

It is generally held that whatever else may be the functions of the lymphoid constituents of Waldeyer's Ring, their protective rôle is the most important of them. But curiously enough, the textbooks seem to have omitted one argument which Nature herself frequently proclaims, and her proclamation may often be read on the walls of the oropharynx — but do we always grasp the meaning of her message?

It must frequently have fallen to our lot to be consulted by young adults whose tonsils were enucleated and adenoids removed when they were young children. What do we sometimes find when they consult us later in life? A hypertrophied, somewhat edematous band of congested mucous membrane corresponding to the so-called "lateral pharyngeal band". Or it may be several large patches of granular pharyngitis on the posterior wall. We may also be surprised to find that—in spite of our perfect enucleation of "the tonsil complete in its capsule" there has appeared a considerable mass of lymphoid tissue in the lower halves of the tonsillar recesses. What do these appearances mean? To me they signify that Nature insists on having some lymphoid tissue in the oro- and nasopharyngeal regions and if it be too radically removed in a child it may be replaced where it is required. That so-called "recurrent" tonsil may not be what our operation left behind, but some of the lymphoid tissue pushed upwards from the side of the lingual tonsil.

If such observations and their explanation be correct, what practical inferences are to be drawn from them? Is our tonsillar surgery sometimes too drastic? Are there any means by which we can foretell when such compensatory developments will occur? I ask these questions because I have seen, and had to operate on chronically inflamed lateral pharyngeal bands as large as an ordinary lead pencil. They had caused much pain when they became acutely inflamed.

One other consideration, and this of ethical bearing. When we meet with these compensatory adjustments in the (possibly) aggrieved patient of a colleague, let us remember that of "Faith, Hope and Charity, the greatest of these is Charity".

SURGERY IN ITS RELATION TO THE TONSILS AS PORTALS OR CARRIERS OF INFECTION.

It is obvious that on this time-limited occasion one can only touch on the general principles which this subject involves. We are all familiar with the acute tonsillitis which often heralds the onset of such acute specific fevers as scarlet fever, acute rheumatic fever and not infrequently diphtheria.

In more stealthy and less dramatic form we know that the tubercle bacilius may invade the tonsillar tissues, slide pass through them and cause a tuberculous adenitis of the cervical glands, and do so without causing any naked eye changes in the tonsil itself.

We have also reason to believe that certain types of arthritis, myositis, neuritis, aural, ocular, cardiac and other distal manifestations of sepsis may be primarily of tonsillar origin.

For a few moments it is only possible to make some brief statements on the most important of these infections.

1. *Diphtheria.* It has been my experience to quickly cure some otherwise intractable diphtheria carriers by enucleation of the tonsils which bacteriological examination revealed the K.L. bacilli in the crypts. One patient had been isolated for six weeks after convalescence, another for nine weeks, although both of them appeared to be in perfect health and were anxious to take up their ordinary work.

2. *Tubercle.* A vast amount of work and investigation has been carried out on the subject of tuberculous infection of the cervical glands by way of the tonsils.

My personal and purely clinical experience supports the contention that true tuberculous infection of the tonsils is not common and occurs in not much more than 5 per cent of the cases. It would appear that enlargement of the tonsils is primarily due to pyogenic organisms and that these bring about the cervical adenitis which later on becomes infected by the tubercle bacillus. This would explain why enucleation of the tonsils is so often followed by the gradual disappearance of the glands in the neck—provided they have not already begun to break down and suppurate.

If such a contention be correct, it follows that the earlier septic tonsils are removed, the better will be the prognosis with regard to the cervical adenitis and of these glands becoming complicated by tuberculous infection. To those interested in the subject, I would refer them to Monographs by Howarth and Gloyne, *Journal of Laryngology and Otology*, Aug., 1924, and by Ritchie Paterson, Ibid., Aug., 1929.

3. Chronic Arthritis. It will generally be conceded that in the absence of definite bony changes around the joint articulations, the removal of septic tonsils will cure or give considerable relief in about 50 per cent of the cases. In this matter I have seen some very gratifying results, but equally disappointing failures, and must confess that, so far, I know of no definite clinical signs on which to base a prognosis. Suffice it to say, that enucleation of septic tonsils seems to promise a good result if the history of the patient shows that an attack of tonsillitis is followed by aggravation of the joint symptoms and also if, in the quiescent periods, the anterior faucial pillars are of a purple-red color.

This appearance generally indicates that the streptococcus viridans is the offending organism.

But we must not forget that disorders of metabolism, might, if corrected, relieve a large percentage of the cases which tonsil enucleation has failed to alleviate. If time permitted, this aspect of the subject might profitably be discussed.

But one of our most important problems seems to lie in the attitude which we as surgeons should adopt in cases of acute rheumatic infection. Let me give you only two examples of the many difficulties which so frequently confront us.

1. A child suffered from a cardiac lesion following immediately upon an attack of acute tonsillitis. After three months' convalescence, I enucleated the tonsils and a fortnight later she was readmitted with some pericarditis, and eventually left the hospital a cardiac invalid.

2. A girl, age 13 years, with enlarged tonsils had suffered from sore throats. Then there supervened an attack of acute rheumatism with mitral disease from which she made a good recovery. Three weeks later the tonsils were enucleated and three days later the temperature rose, and a dangerous and nearly fatal carditis and pericarditis rapidly developed.

Now arises the problem—were these patients directly inoculated by the micrococcus through the raw operated surfaces while their susceptibility to infection was still high?

I quote these two cases from the Lettsomian Lectures "On Rheumatic Heart Disease in Childhood", Trans. Med. Soc., London, 1928, and delivered by my friend and hospital colleague, Dr. F. J. Poynton. As you may know, he and Dr. Paine, in 1899 and in the earlier years of this century, were the first to establish, by animal experimentation and clinical observation, the streptococcal origin of rheumatic infection.

The cases I have cited prompt two questions at least.

1. When do the tonsils lose their protective function and become a source of danger?

2. How are we to determine when convalescence from an attack of acute rheumatism is so complete that we may safely remove diseased tonsils in order to prevent or minimize the chances of a further attack.

I hope some of you will be able to answer these questions.

Poyton asks (loc. cit.) a question which we must often have asked ourselves, *viz.*,

"A child has an attack of rheumatism with evidence of tonsillitis; there is apparent recovery and the tonsils seem healthy and the tonsillar glands are not enlarged, or only to a slight degree. Ought we not to advise their removal as a precautionary measure? If we feel we should do so, should we operate at the end of the acute illness, or wait until the child has made a thorough convalescence?"

It would be very interesting to hear your experience as to the occurrence of first attacks of rheumatism in children who have already had their tonsils enucleated. If it be frequent, then we must be more careful in the selection of cases for surgical treatment.

MALIGNANT DISEASE OF THE TONSILS.

Primary carcinoma and sarcoma are the two commonest forms, and I only refer to these lesions because on a few occasions patients have presented themselves with a mass of hard glands behind the angle of the jaw and no primary lesion could be seen in the throat or nasopharynx. But on enucleation of the tonsil and microscopic examination of the sections, the small and unsuspected primary growth was found. I will show you a slide exemplifying this somewhat uncommon condition.

An analagous but more obvious condition is sometimes seen in the tongue, *viz.*, a small primary growth and a large mass of glands or, conversely, a large fungating ulcer with very small metastases in the cervical nodes.

With regard to the treatment of the primary growth in the tonsil, radiotherpy seems to have completely ousted cutting operations and diathermy. One need scarcely say that small doses of the screened element, or of its emanations, are inserted into and around the growing margin of the tumor and left for five to eight days. Infected glands in the neck are removed by a block dissection either before or after radiation of the tonsil lesion, and the affected triangles in the neck are given further exposures from time to time.

The increasing frequency of the results of such treatment must surely be amongst the triumphs of modern medicine.

POST-OPERATIVE COMPLICATIONS OF TONSILLECTOMY.

Hemorrhage. An experience of 33 years seems to have proved to me that this is the most frequent accident following removal of the tonsils and the one most feared by the patient if he or she be an adult, or by the parents in the case of their child. If one visualizes for a moment the anxiety which such a complication may cause, the possible necessity for a second anesthetic in order to secure one or more bleeding points, the alarming degree of anemia which may quickly result, and the prolonged convalescence to normal health—we may well ask ourselves the question: "Do we always sufficiently prepare our patients for this operation, and in carrying it out do we endeavor by proper surgical measures to minimize the risk of postoperative bleeding?"

To go into lengthy answers to these questions is impossible and I can only briefly state my own views and practice. For some 15 years, I have enucleated all tonsils—in private practice and in hospital—by dissection. Previous to that period I used the guillotine method as introduced by Whillis, Pybus and Sluder, and by keeping a careful record of all cases from infancy to old age—my oldest patient was 72 years, who suffered from three to four quinsies a year—my cases of bleeding which required active intervention amounted to about 5 per cent. Since I employed dissection they have been reduced to 1 per cent.

I believe the reduction may be explained in two ways.

1. By the well known physiological fact that the torn or lacerated end of an artery contracts and retracts more readily than if it be cut or divided by a more or less sharp instrument.

2. In every patient, from the youngest to the oldest, I ligature the descending branch of the posterior palatine artery and, if necessary, the tonsillar branch of the facial in the lower region of the tonsil recess.

Furthermore, no patient is allowed to leave the operation table until the tonsil beds are dry.

The time required for such operative details is rarely more than 10 to 12 minutes. Compared with the guillotine method, dissection takes longer, but stopping all bleeding demands less time, so what we "lose on the swings, we can make up on the roundabouts".

For years I have preached the advisability of ligaturing, but at first my voice was as of one crying in the wilderness; today, I am

glad to say that this practice is becoming more universal, at any rate in England. To place a ligature securely around an actively bleeding vessel requires possibly five to six seconds, and the operator leaves the home or hospital with his mind free from any anxiety. When surgeons write in the journals or tell me they check operative hemorrhage by pressure with gauze swabs, I would ask them two questions.

1. "Why do you do for the tonsil recesses what you would not do in the case of a bleeding artery in any other accessible part of the body?"

2. "How do you know that there will not be some postoperative vomiting, crying or restlessness, and that the strain of these will not reopen the unsecured end of a small artery?"

I know it requires considerable practice and some little dexterity to recognize and tie the chief arteries which supply the tonsils, but surely it is our bare duty as experts to attain some particular skill in our craft, otherwise wherein lies the *raison d'être* of our calling? Nevertheless, however careful we may be in the preparation of our patients and in the technique we adopt, an occasional postoperative hemorrhage will prove that we are but human and our methods fallible. About 12 months ago, I enucleated the fibrous tonsils of a man who was subject to recurrent quinsies. The hemorrhage was so excessive that I ligatured seven bleeding points and the tonsil beds were dry when he left the operating theatre. Four hours later, hemorrhage recurred and before I could get to him, he was pulseless at the wrist, exsanguinated and begging in a whisper for more air. Intravenous saline injections, stimulants and the removal of clots from the tonsil recesses stopped the bleeding, and he recovered. Fortunately, such experiences are rare and they should continue to be so, but only if we employ the first rule of surgery in checking arterial hemorrhage, *viz.*, to seek for the bleeding point and ligature it.

I know no other method by which we can discharge the responsibility of doing our best for the safety and sanctity of a life entrusted to our care.

PULMONARY COMPLICATIONS AFTER TONSIL OPERATIONS

Of these it has been my good fortune to have had practically no experience in my own practice, nor have I seen such cases in consultation with my colleagues. Sir St. Clair Thomson makes a similar personal statement in the last edition of his book on Diseases of the Nose and Throat.

It is therefore impossible for me to discuss a complication from which we seem to be relatively immune in England.

From your literature on the subject I gather that you consider that severe pulmonary complications may arise from direct inhalation of septic material which is liberated at the time of operation on the tonsils or from septic thrombosis of the veins in their recesses which lead to the formation of pulmonary emboli.

I do not know in which position you place your patients during enucleation; ours generally have the head extended by means of a sandbag under the shoulders, so that all blood and secretions pass into the nasopharynx and cannot get into the lower air passages. Again, we never adopt the sitting or semiprone positions. No pillow is allowed in the bed until the patient has recovered the cough and swallowing reflexes.

Finally, obvious dental sepsis is treated before the tonsils are operated on.

And now, Mr. President and Gentlemen, I have only to thank you for so patiently listening to me while I have tried to discharge the honorable task you invited me to undertake.

Like the song of Nanki Poo in the Mikado, my contribution to your proceedings has been, I fear, "a thing of shreds and patches", little experiences and thumbnail sketches drawn from a pilgrimage of 33 years, but if any of them have interested you or stirred your imagination I shall indeed take away with me my most abundant and satisfactory reward.

PYEMIA FOLLOWING THE ANGINAS.*

DR. DAVID NUSSBAUM, Philadelphia.

To further emphasize that the tonsils are a source of infection for the human organism would appear like carrying coal to Newcastle.

There is today hardly any dispute as to their responsibility for 80 per cent of all acute and chronic rheumatic disorders; for the very large number of kidney infections; for septic endocarditis and chorea.

But we also encounter—in the trail of an acute attack of tonsillitis—quite an imposing collection of very severe and treacherous cases, winding up with an appalling mortality, the records of which rarely reach the general man, but remain tucked away in the pages of our special literature.

No wonder, then, that we are awe stricken and perplexed in the presence of these cases, generally too far gone when they reach the operating table.

Should, therefore, even one patient be helped at the opportune time and his life saved, any amount of discussion would be justifiable.

PATHOGENESIS.

The tonsils are analogous in texture and function to the rest of the lymphoid tissue, densely scattered within the mucosa of the adjoining pharyngeal structures; subject, consequently, to the same injurious influences that affect the entire lymphoid ring.

This fact has induced many investigators to suggest the generic name of "angina" for all forms of acute infectious diseases originating in the mucous membrane of the naso-, oro- and hypopharynx; from the simple catarrhal to the exudative, phlegmonous and suppurative inflammation, including Ludwig's and Vincent's angina. The term "tonsillitis" is applied to infections originating in and limited to the tonsil proper.

The exciting factor is the aerobic group of strepto-, staphylo- and pneumococcic bacteria, always found in acute cases, and alongside with them a goodly number of pathogenic strains, like the hemoliticus, influenza and diphtheria bacilli; fortunately, in the harmless stage of parasitism.

*Read before the Philadelphia Laryngological Society, April 2, 1929.
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication April 24, 1929.

Generally speaking, the streptococci are by far the more malignant, due to their inherent tendency to spread over wide areas and to cause metastases in the joints and muscles; rarely in the lungs.

A related anaerobic strain, the streptococcus putrificus, is mostly responsible for lung abscesses and purulent pleurisy, with fatal results in the short space of between 8-14 days.

It should be borne in mind, however, that it is not the degree of bacterial virulence alone that shapes the future course and determines the severity of the disease. This has been proven by R. Waldapfel, who injected into his cubital vein 20 c.c. of blood from a tonsillitis patient, who was running a temperature of 103.1°. One day later, he rubbed into his tonsils, after previous scarification, a smear from the same patient's throat, without any bad effect following either experiment.

There are, indeed, a multitude of other factors, like chilling, dust, nasal operative traumatism, seasonal changes, which play just as effective a part as the germs themselves, by reason of their tendency to undermine the resistance of the befallen individual.

This explains why, instinctively, the layity attaches so much importance to climatic changes with their correlated effect upon the body temperature.

Exposure to chilly and damp weather, wet feet, will actually induce "colds", probably by disturbing the circulation, interfering with the germicidal properties of the blood and otherwise disrupting the biologic equilibrium of the individual.

In what other manner could we explain the prevalence of anginas in winter and spring?

The portal of entrance for local as well as general infection is still a debatable subject. A great many observers believe it to be the palatine tonsils, while quite many others place it in the pharyngeal tonsil (adenoids); and this seems to hold true in the case of children, who make a wonderful recovery after a simple adenectomy.

There is still another opinion that classifies all complications in connection with an acute attack of tonsillitis as a generalized infectious disease, with the involvement of the pharyngeal tissues as a mere conspicuous local manifestation—a symptom among other symptoms—and nothing else.

This theory is frequently advanced but not generally accepted, although in a majority of instances it is next to impossible to fasten —scientifically speaking—on the poor tonsils the multitude of sins we are prone to attribute to them.

The tonsillar infection may stay localized or spread to the tissues immediately contiguous, infiltrating the superficial layers of the mucosa; or it trespasses the limits of the primary region and infects the several groups of cervical lymph glands that drain it.

Should the inflammatory process, however, penetrate deeper into the submucous strata, it inevitably leads to phlegmonous changes in the peritonsillar and parapharyngeal spaces, and advances through the latter into the mediastinum and cranial cavity.

The lymph spaces are frequently used as a pathway by the bacteria, giving rise to lymphadenitis of the upper cervical group, and in severe cases the germs filter through this first line to the lower end of the gland chain, around the clavicle, and even to the peritracheal and periesophageal glandular cluster.

In scarlet fever and serious forms of grippe and influenza the infection shows a marked tendency to spread far beyond the tonsils, into the pharynx, larynx and trachea, affecting, as in erysipelas, the uppermost layers of the mucous membrane, which is loaded with streptococci, is bathed in fibrinous and purulent transudate, becomes puffed up and edematous and threatens with stenosis and actually closes up the larynx.

As a contributing factor, of course, there is the breaking down of body resistance, the total lack of fighting ability and no strength to stop the relentless onrush of the disease.

During acute attacks of tonsillitis the peritonsillar tissues may become sympathetically involved by collateral edema, which recedes simultaneously with the subsiding of the angina; or the inflammation is progressing further into the loose connective tissue and recesses surrounding the tonsils and gives rise to a genuine peritonsillar abscess.

The latter, however, halts at the constrictor pharyngis muscle, the outer boundary of the peritonsillar niche.

The causative sequence is not so evident in cases of chronic diseased tonsils with their deceiving, healthy, clinical appearance; unless it be borne in mind that, in spite of their good looks, they harbor within and around them numerous pus foci, filled with pathogenic germs, ready to flare up at the slightest provocation by a most trivial cold.

They are, thus, a never-ending source for any number of recurrent peritonsillar abscesses and the proof of it is that they disappear with the removal of the tonsils. Not to be forgotten are bad teeth and diseased sinuses, which contribute their share.

ANATOMY.

Externally to the superior constrictor muscles and the very thin fascia pharyngea which lines and closes up the pharyngeal tube, lies a space which covers an area of about 2 sq. c.m. wide. It is called the "parapharyngeal or pharyngomaxillary region".

It is roughly enclosed by the above-mentioned muscle, the vertical branch of the mandible, the parotid gland and the transverse process of the atlas, with its prevertebral fascia. For the size of its area, it houses the most powerful collection of nerves and blood vessels found anywhere in the body (the carotids, the jugular vein, vagus, hypoglossus, glossopharyngeus, accessorius and the sympathetic).

It also contains many muscles, fat, loose connective tissue, body fluid and some of the upper cervical lymph glands draining the tonsillar region.

It merges sidewards with the retropharyngeal space (situated between the pharyngeal tube and the vertebral column); it communicates through the jugular foramen, carotic canal and foramen lacerum with the cranium and continues downwards, merging with the sheath of the large vessels, into the mediastinum.

The symptoms referable to an inflammation of this region are very variable as to character and degree; submaxillary adenitis, ankylostoma, brawny swelling of neck, associated with deep suppuration and edema of the larynx, parotitis, neuralgia of the trigeminal branches, otalgia; impaired, exceedingly painful swallowing, and muffled speech —they may all be present or not, very severe and life-threatening, or very mild and unnoticeable.

The infection rarely stops at the submaxillary, but involves the more deeply located cervical glands of this and the retropharyngeal areas and those around the carotid artery.

Retropharyngeal adenitis is mostly observed in children, it is seldom absent in scarlet fever, and is common occurrence in those with recurrent attacks of acute rhinitis and rhinopharyngitis.

Since, in a great number of cases, the adenitis ripens into an abscess, it is advisable to follow the golden rule, to always palpate a child's throat and to guard against a fatal blunder. These abscesses are generally found behind the posterior pillars, also in the median line and lown down in the hypopharynx; while they are rare in adults, they are nevertheless met with.

Even in the presence of very virulent bacteria, as long as the resisting powers of the individual are up to the mark, the infection will be combated and alterative changes will take place in the involved

tissues in the form of exudate, infiltration and proliferation, with the end-result of a walling off of the phlegmonous exudate.

If, on the other hand, the fighting power of the patient is low, or the bacterial strain very virulent, and if by chance they join forces, then no barrier can be erected by the exhausted organism and all flood gates remain wide open to the large blood vessel area, with the imminent danger of thrombosis of the veins and erosion of the carotids (of which several hundred cases are on record).

This is a grave and serious danger, but it assumes the proportions of a calamity when the infection gravitates towards and into the cranial or mediastinal cavity.

The transmission mechanism of the tonsillar infection into the circulation is a controversial question as yet. One group of investigators claim the exclusive pathway of the lymphatics, by pointing to the seldom missing glandular involvement.

The seat of these glands is upon and around the large vessels (jugular, carotids) and it is obvious, that when these glands break down and suppurate, they should cause thrombophlebitis or thrombosis of the adjacent vessel. The phlebitis remains either stationary and subsides, or it expands by continuity towards the mediastinum and cranium.

Another group of observers lay stress on the ever-present thrombophlebitis of the retrotonsillar veins, which may infect the bloodstream either by themselves or through the medium of the jugular vein into which the inflammaiton is expanding by continuity.

There are on record a substantial number of severe cases of sepsis and pyemia without any demonstrable retrotonsillar suppuration, adenitis or thrombophlebitis; where the germs manage to break directly into the vascular system, disseminating pyogenic emboli all over the body and causing any number of metastatic abscesses.

In acute and acutely exacerbated chronic tonsillitis, therefore, when thrombotic masses glut the small and large adjoining blood vessels (palatine, pterygoid, anterior, posterior and common facial and the internal jugular vein) and softened, germ-laden particles become detached and are swept into the bloodstream, the connection between tonsils and sepsis or pyemia is self-evident.

In chronic inflamed tonsils the issue is not as clear-cut as this; here we may only presume—and quite probably it is so—that a continuous flow of toxins and pathogenic germs finds its way—from clinically undetermined pus foci into the blood circulation, maintaining a low-grade systemic infection. It manifests itself by a most

varied and peculiar symptomatology—sailing under the overworked label of "focal infection".

Although it is impossible to support this presumption with positive bacteriological findings, as repeated blood cultures may prove exasperatingly sterile, or to trace the course and entrance point of the micro-organism into the circulation, it is nevertheless an unescapable conclusion in acute cases where cause and effect follow on the heels of each other, and a most probable condition in chronic tonsillar disorders.

The route of infection into the cranial cavity is by way of a thrombus of the jugular vein and lateral sinuses or by means of a phlebitis of the several intercommunicating pharyngeal venous plexuses (retrotonsillar, pterygoid, around jugular foramen), extending directly into the cavernous sinuses.

The contamination of the thoracic cavity takes place when the angina descends alongside the sheath of the vessels and nerve trunks housed in the pharyngomaxillary space.

The sequence of events leading to the clinical picture of metastatic pyemia would be as follows: acute tonsillitis, retrotonsillar adenitis and suppuration, phlegmon of neck, edema and phlegmon of larynx, phlebitis of pharyngeal venous plexuses, thrombosis of jugular vein and cavernous sinus, extradural and brain abscesses, encephalitis and meningitis.

DIAGNOSIS.

Acute tonsillitis and peritonsillar abscesses are in the great majority of cases easily recognized, but the correct diagnosis of the pathology of the parapharyngeal space meets with great difficulties; more so when complicated by peritonsillitis, parotitis or posterior nasal inflammatory conditions. All of these are associated with a circumscribed or diffuse adenitis of mild or severe character, which makes the palpation of the deeper structures exceedingly difficult, if not totally impossible.

Thrombosis of the jugular vein may become overshadowed by the existence of a very painful cervical adenitis (submaxillary), or the original infection may have subsided to such a degree as to offer no lead to a diseased vein and yet its existence is an unwelcome surprise on the operating table.

The only reliable finding is, after all, the palpation of a tender, cord-like vessel, although the absence of this symptom excludes by no means the presence of a thrombus. This possible complication should always be kept in mind and looked for, and it will be more often diagnosed than is actually the case.

TREATMENT.

The treatment is to follow along surgical lines and pus evacuated wherever found. There is still a dispute concerning the advisability of enucleating the tonsils in the florid stage of peritonsillar inflammation; but, given a case with no yield of pus in spite of several successive incisions, the tonsil better be removed immediately, mostly when proven to our satisfaction that it is beyond doubt the primary lesion of a generalized infection.

Following the removal of the cause, particular attention should be paid to the jugular vein, and in the presence of a probable or definitely established thrombus, it should be approached in the same sitting, without the slightest hesitation.

The patient is given the benefit of several days of digitalis administration, and two hours before operation an intravenous injection of 50 c.c. of a 20 per cent glucose solution. Local anesthesia is to be used.

The entire jugular field, including its tributaries (tonsillar, facial, etc.), is exposed as far back as the tonsils, if such a contingency arises, the vessel and its sheath freed from adhesions and diseased or suppurating lymph glands and carefully searched for thrombotic masses.

When such are found, ligatures, preferably double, should be secured in the healthy wall portions, the vessel opened and the clots cleaned out until a free flow of blood is established. Necrosed areas in the wall are completely excised and all pus pockets around the vessel and nerve field thoroughly drained.

In this manner cause and effect are eliminated simultaneously, the system freed of pus collections and their sequellae, the adjoining tissues protected from further contamination and destruction, and the patient helped along to a gradual regaining of health.

Several objections have been raised against this operation, as, for instance: *a.* the number of patients saved is not larger than those not operated upon; *b.* the jugular vein is not the only route selected by the bacteria, there being other channels that accomplish the same purpose (lymph system); *c.* danger of stirring up the quiscent thrombus to new life and the induction of a retrograde spreading of same to every area of the body.

But notwithstanding these shortcomings it is at present the only hopeful chance we have to offer our patients, provided, we do recognize the situation in due time and do not temporize with all sorts of dyes and foreign proteins until the patient is hopelessly lost; we may

fare, at least, just as well as in septicemia of otitic origin and not assume a fatalistic attitude.

To arrest the descent of the infection into the mediastinum, the following procedure has been suggested and performed, namely: collar-like incision over sternal notch, exposure of the sheath of the vessels up to the clavicle, severance of all adhesions, drainage of pus, if found, and packing with iodoform gauze of the thoracic opening in back of the jugular notch, similar to corking up the neck of a bottle.

Up to 1927 not more than 58 cases of post-tonsillar pyemia have been reported in the literature, native and foreign, with only 13 recoveries following ligation and total excision of the jugular vein; two of these cures are recorded by American surgeons.

Lately, more and more cases of this type are published here and abroad, with a larger percentage of lives saved; due, in all probability, to a better understanding and more rational treatment.

So, Claus, of Berlin, is able to report recently three successive recoveries by means of excision of thrombosed veins from the base of the skull down to the clavicle and from the jugular bulb all the way to the innominate vein, with simultaneous enucleation of the tonsils. From 20 such patients within the last year he lost 11 and saved nine.

On the other hand, Frederick Hill, of Waterville, Me., reports only as recently as last month a fatal case of peritonitis, following a hidden, none too severe peritonsillar abscess, 11 days after the onset of an acute attack of tonsillitis and sore throat.

The greatest obstacle to the recognition of the seriousness of these cases, before it is too late, is the fact that the pyemic or septic complications appear much later on the scene, at a time when there is almost no vestige left of the previous acute infection and all looks clinically well.

If misfortune is lurking, the other pathognomonic symptoms (chills, high fever, locked jaws, etc.) may also be absent, or hardly noticeable, and the patient is treated expectantly for a harmless lymphadenitis, which is always present, until the storm breaks loose from a clear sky.

Therefore, a lingering adenitis long after the acute angina has subsided, if associated with severe constitutional disturbance, or even general malaise, gives cause for anxiety and worriment, bears the closest watch and needs the trained eye and intuition of the specialist, who is in a better position, as matters go, to handle these cases.

It is rather a kind act of fate, that the fulminant cases are the exception to the rule, and many of these phlegmonous anginas run a

much milder course, regardless of the presence of chills, high temperature, cervical lymphadenitis, peritonsillitis and a palpable, tender, cord-like jugular vein; they improve by a simple tonsillectomy or lancing of the peritonsillar abscess.

SUMMARY.

A streptococcic sore throat is always a cause for alarm, because the infection does not stay localized but spreads beyond the tonsillar region, as follows:

a. To the contiguous tissues, causing peritonsillar and parapharyngeal suppuration.

b. To the blood vessels, giving rise to phlebitis and thrombosis. This is effected by extension from the smaller (tonsillar) to the larger (jugular, lateral sinus, cavernous sinus) veins in every direction; or by adherent infected and suppurating lymph glands; or by both ways. Detached, infected thrombi may break into the circulation and result in scattered metastatic abscesses.

c. The bacteria invade directly the vascular system—pure hematogenous infection—and we deal with either a bacteremia, with numerous pyogenic emboli, due to glutting of the capillaries by microorganisms; or with a toxemia of pure septic character. Persistent lymphadenitis in spite of a subsided acute angina is a red danger signal.

Treatment is surgical at the earliest possible moment, aiming at the tonsils themselves and at their, so to say, infected hinterland by enucleation, exposure of the venous field, ligation, removing of clots and total excision of infected vessels, in addition to drainage of detected pus collections.

Local anesthesia is imperative to spare the toxic patient the burden of another poisonous load of general narcosis. The operative procedure fails in septic cases with marked toxemia and absence of thrombosis.

May I be permitted to say in conclusion, with all due modesty, that surely no one is able to foretell which cases will run a smooth and which a rough, stormy and fatal course; therefore, any suspicious case of streptococcic sore throat is entitled to expert treatment at the hands of the trained otolaryngologist, at as early a date as possible.

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LATERAL SINUS THROMBOSIS, WITH REPORT OF TWO CASES.*

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Introduction. In a review of the recent literature on lateral sinus thrombosis it was found that this subject has been thoroughly covered from almost every angle. Many articles have been written which consider only the pathological characteristics; others have carefully covered the differential diagnosis; many others have been devoted to the question of treatment; and still more have emphasized only the clinical picture, with presentation of cases. In a subject of this kind, about which so much has been published, it is difficult to add anything new to the information now available. The author has therefore attempted only a brief resumé of the statistical data concerning the incidence of lateral sinus thrombosis and added two more cases to the already large number of published cases of this kind.

Incidence. Statistics on the involvement of the lateral sinus following mastoiditis vary greatly. It is stated by some writers to be more common in those cases due to chronic otitis media and mastoiditis, while others claim it is seen oftener in acute infections. Fraser's[1] statistics, which are in accord with those of the majority of European observers, show that 75 per cent of cases of lateral sinus thrombosis follow the chronic infections. On the other hand, Atkins[2] reported 40 cases of sinus thrombosis, of which 75 per cent followed acute suppurative otitis media, while the remainder occurred in chronic suppurations. His series also showed that 70 per cent of the cases were in males and 85 per cent occurred on the right side.

Don Campbell[3] states that statistics show the right lateral sinus is larger than the left and its relationship to the mastoid cells is such that it is rendered more liable to infection than the left in the proportion of 3:2. However, Rugg[4], in a study of 422 otogenous brain complications, has found these complications as a whole to be equally divided as regards the two sides.

It has been found that there is also some difference of opinion on the percentage of cases of sinus thrombosis that accompany or follow mastoiditis. White[5] stated that the fifty-first annual report of the Manhattan Eye, Ear and Throat Hospital gave a series of 566 cases

*Read before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, Feb. 20, 1929. From the Department of Otolaryngology of the University of Pennsylvania.

of acute and 22 of chronic mastoiditis, with only 11 cases, or 1.8 per cent, of sinus thrombosis.

Kopetsky's[6] figures show a much higher incidence of sinus thrombosis. He reported 25 cases, or 20 per cent, in 126 cases of coalescent mastoiditis, and six cases, or 30 per cent, in 20 patients with acute coalescent mastoiditis superimposed on chronic purulent otitis media. In his analysis of 44 cases of hemorrhagic mastoiditis he found 15 cases of sinus thrombosis, or 34 per cent.

Most authors, however, agree that lateral sinus thrombosis occurs in from 3 to 6 per cent of the operated cases of mastoiditis. Naftzger[7] reports six cases in 192, 3.12 per cent, and quotes Hill as having seven cases in 166, 4.21 per cent; Downey, five cases in 79, 6.32 per cent; Gerber, 25 cases in 524, 4.77 per cent; and the Massachusetts Charitable Eye and Ear Infirmary, 19 cases in 497, 3.82 per cent. Statistics from the Mayo Clinic[8] show that during a period of five years there were found 19 cases in over 500, 3.8 per cent. Yerger[9] reported a series of 34 cases of sigmoid sinus thrombosis, which was 3 per cent of the cases of suppurative otitis media.

CASE REPORTS.

The following two cases of lateral sinus thrombosis, while typical in many respects, may possibly be of unusual interest because of the coincidental occurrence of other complications.

Case 1: A. S., female, age 19 years, was admitted to the University Hospital on Feb. 15, 1928, with pain in the right ear, chills and fever.

The history of the present illness showed that about Jan. 20, 1928, the patient had a chill, followed by a temperature of 103° F., with pain over the frontal and occipital regions and in the stomach. The ear was not painful and discharged only slightly. Since then she has had at least one chill, sometimes two, and occasionally three, a day. Her condition was not definitely diagnosed and her doctors apparently were suspicious of typhoid fever, tuberculosis of the lungs and meningitis. The temperature had been running up to 102° and 103° F. in the afternoon.

Four days before admittance a swelling was noted below the angle of the right mandible, and yesterday there appeared an intensely tender swelling behind the right ear. A slight discharge from the right ear had been present for one week.

There had been no stiffness of the neck. For a time she had had considerable cough, with expectoration. Examination of the sputum was negative for the tubercle bacillus. Blood tests for typhoid fever were negative. The urine had also been reported negative. At the

beginning of her illness she had lower abdominal pain for 8 or 10 days and had vomited four or five times.

The previous medical history revealed that at the age of 2 years, the patient had had scarlet fever, which left a discharging right ear. Since that time there had been an intermittent discharge from that ear and the patient had been slightly deaf.

The family and social history were negative.

The physical examination on admittance showed a poorly nourished young woman, evidently acutely and gravely ill. There was a tender swelling of the right side of the neck below the angle of the jaw extending up behind the ear. Examination of the ears showed a small amount of a thick, foul, purulent discharge in both canals. Both drums were practically absent and there was present considerable red granulation-like tissue in the middle ear of both sides, with a little thick pus overlying it, which, in the right ear, was pulsating. There was no bulging of the canal wall.

The lungs showed a few rales over the left chest anteriorly. The heart rate was rapid but of regular rhythm. No murmurs heard. The abdomen was negative.

The blood pressure was 110 systolic. The leukocyte count was 25,800.

On the next day neurological examination showed the cranial nerves negative except for bilateral impairment of hearing, the tendon reflexes normal and equal, no Babinski or ankleclonis, no demonstrable weakness of muscles or extremities and no spontaneous nystagmus.

Examination of the eyes by Dr. A. G. Fewell showed the pupils equally and evenly dilated. The discs showed well marked choking, measuring 6 diopeters, with numerous flame-shaped hemorrhages and exudates about the discs. The veins were enlarged and tortuous.

The blood count on the day after admittance gave 20,400 leukocytes and 57 per cent hemoglobin. Lumbar puncture by Dr. W. J. Gardner gave a spinal fluid pressure of 65 c.m. of water. One-half cubic centimeter of clear fluid was withdrawn, which gave a leukocyte cell count of four. Attempt to make a Tobey test was unsatisfactory because of the extremely high pressure of the spinal fluid.

Medical consultation revealed the patient to be in an extremely septic condition, but no frank meningitic symptoms. Lungs and heart were negative. Urine showed a trace of albumin and many cellular casts. The high temperature for the day was 102.4° F. and the low was 98° F.

Roentgen ray of the mastoid processes by Dr. H. K. Pancoast showed a large abscess cavity on the right side with breaking down of all the cell walls that encroached on the lateral sinus. The left mastoid showed sclerosis of some of the cells, probably due to an old chronic mastoiditis.

Operation. After consideration of the results of the examinations, a diagnosis of lateral sinus thrombosis was made. Operation the same day, on Feb. 16, revealed the following: After incision through the overlying tissue the mastoid cortex appeared roughened. The cortex was extremely thin and only slight pressure of the gouge caused the instrument to fall through into a rather large abscess cavity filled with pus and granulating material. The cell walls were entirely broken down, as was also the plate overlying the sigmoid sinus. The sinus lay exposed in the wound for a length of about an inch and was covered with a thick layer of granulations. On palpation it was of firm consistency and gave a marked pulsation.

The mastoid cavity was cleared out, a large opening made into the antrum and the dura exposed through the tegmen antri. The dura was normal in appearance, so was left alone. The tip of the mastoid was found broken through and on pressure on the swelling below the angle of the jaw a large amount of foul pus was forced into the mastoid cavity. A hypodermic needle was inserted into the lateral sinus, but nothing could be withdrawn. After further exposure of the sinus to slightly beyond the knee, an incision was made lengthwise in it and the sinus found to be completely obliterated by a firm clot in the early stages of organization. At this stage further mastoid manipulation was delayed until Dr. Ferguson had ligated and cut the right jugular vein. There was no thrombosis of the vein and it bled freely before ligation. The clot in the lateral sinus was then removed by means of a curved curette, which was inserted up beyond the knee into the transverse portion of the sinus as far as the torcular. It was only after considerable scraping in this portion of the sinus that free bleeding was obtained and it gave the impression of a clot well back in the transverse portion probably extending to the torcular. In the lower end of the sinus the clot was found to extend at least to the jugular bulb and after removal of much of it a small amount of bleeding from that end was obtained.

Drainage through the tip of the mastoid into the cervical region of the neck was next instituted and two counter-openings made in the neck, with through and through rubber tube drainage. The mastoid wound was left open. Cultures of the contents of the lateral sinus showed, after four days, only diphtheroids (probably contamination).

Culture of the pus in the mastoid showed bacillus proteus mirabilis. Culture of the spinal fluid showed diphtheroids.

On the day following the operation the mental and general condition of the patient were somewhat better. She was given a blood transfusion of 250 c.c. The high temperature was 103.8° F. and low was 98.8° F.

On the next day, Feb. 18, spinal puncture revealed a pressure of 31 c.m. of water. Unilateral compression of the jugular gave an identical response on each side of 40 c.m. of water. Bilateral compression for 10 seconds caused a rise to 44 c.m. Pressure on the facial veins bilaterally gave a rise of 2 to 3 c.m. Dr. W. J. Gardner gave the following opinion of the results of these tests: "These responses may mean that there is a bilateral lateral sinus thrombosis and that a collateral venous return is being established in the external jugular and spinal veins. I have not sufficient confidence in this interpretation to advocate operation on the other lateral sinus as yet."

One-half cubic centimeter of spinal fluid was withdrawn and the cell count was 27 leukocytes.

A blood culture was made and showed a pure growth of nonhemolytic staphylococcus albus on glucose broth medium only after nine days of incubation. Cultures on plain broth and agar plates showed no growth. The temperature varied between 103° and 97.6° F.

On Feb. 20, four days after operation, another incision was made in the neck below the angle of the jaw for better drainage, as the other openings were not taking care of the very large amount of foul spus from this region. Another blood transfusion of 210 c.c. was given, and a blood culture taken, which showed after eight days of incubation a growth of nonhemolytic staphylococcus albus, in glucose broth medium. Neurologic examination was negative except for a mild horizontal nystagmus on looking to the right. The morning temperature was 98.8° F. and the evening temperature 100.2° F.

On Feb. 24, a week after operation, the jugular wound showed signs of infection and was opened widely to allow of free drainage. Dr. Fewell reported the swelling of the optic discs unchanged.

Two days later the spinal fluid pressure was found to have reduced to 15 c.m. of water. Another blood transfusion was given. The temperature ranged from 102° to 98.6° F. The next day a new pocket of pus was found under the subcutaneous tissues, about 2 inches behind the upper end of the mastoid incision. An opening was made to communicate with the mastoid wound.

On March 1, a large abscess was opened, which occupied the lower end of the jugular ligation wound and extended deep into the neck

under the sternomastoid. Another blood transfusion of 250 c.c. was made. It was noted that many small pustules had developed over the back and sacral region and a large one over the right scapular region. Blood culture taken today was negative after eight days' incubation. The temperature for the past few days had varied between 101° and 98° F.

Three days later the patient started coughing and bringing up a mucopurulent expectoration. Examination of the chest showed signs rather suspicious of a pneumonia in the right lower chest. The eye grounds still showed 5 diopters of choking of the discs but no hemorrhages or exudates.

On March 7, 20 days after operation, an examination was made by Dr. C. H. Frazier. He said he was reasonably convinced that there was no indication for exploring either for cerebellar or cerebral abscess. He thought the suggestion of a bilateral lateral sinus thrombosis earlier made by Dr. Gardner was not an improbable one. Further neurologic examination showed normal reflexes and sensation and no nystagmus. Blood count showed 16,300 leukocytes and 42 per cent hemoglobin. The temperature ranged from 102.6° to 100.4° F.

X-ray examination of the chest on the following day showed a slight shadow at the right base posteriorly, probably due to a localized area of pneumonitis.

Examination the next day showed that the pustular eruption over the back had increased somewhat and the lesion over the right shoulder blade had developed into a very large carbuncle, with considerable necrosis of tissue and a profuse purulent discharge. The blood transfusions had been continued every second day, the amount given ranging between 250 c.c. and 270 c.c.

During the next few days the condition of the patient changed but little, the temperature running between about 102° and 98.6° F. She seemed bright, but was extremely weak. The carbuncle on the back became enormous in size and drained profusely. She continued to have an occasional chill and vomited once or twice nearly every day.

On March 12, 25 days after operation, the high temperature was 105° F. An abscess of the buttocks was opened and about one-half ounce of pus expressed from it, and about an equal amount from the large necrotic area over the right scapula. On examination of the chest there was a very loud grating noise heard over most of the chest, as well as over the heart and sternum synchronous with respiration. It was thought to be of pleural origin. She had been expectorating a bloody sputum lately, but this might have come from

the nose as she had had mild epistaxis several times. She complained today of considerable pain in the presternal and precardial region.

On the following day, while having a thermolite treatment of her back, the patient became quite cyanosed and dyspneic. After a change of position she improved. She was sent down for a Roentgen ray of her chest and the report showed some clouding of both bases and some mottling throughout the lungs, the appearance being that of bronchopneumonia. Several hours later, however, she again suddenly became very dyspneic and cyanotic, with extremely rapid and weak pulse, and died within a few minutes. All efforts to obtain a post-mortem examination were futile.

Comment. This patient presented several interesting features from a diagnostic standpoint. She had been treated at home for over three weeks before admission to the hospital for typhoid fever and tuberculosis. Attention was called to the ears as a possible cause of her condition only after a mastoid abscess had grown to a point where it had broken through the tip and spread into the deep tissues of the neck. It seems very probable that at the beginning of her illness, when she first developed chills and fever, she had even then a lateral sinus phlebitis or thrombosis with septicemia. At the time of operation the thrombus was so extensive and showed such signs of organization that it must have been present in the vein for a considerable time.

After admittance to the hospital and the results of the Roentgen ray examination, laboratory tests and spinal puncture could be correlated, it was not a question so much of a diagnosis of lateral sinus thrombosis as it was of the extent of this involvement. The Tobey test indicated a possible bilateral sinus thrombosis, which was never definitely disproved or verified. The operation showed that the thrombosis extended well back into the transverse portion of the lateral sinus and it is very possible that there was extention of this process past the torcular Herophili into the lateral sinus of the opposite side. If the patient had lived longer and still had shown the signs of general sepsis it is very likely we would have investigated the opposite lateral sinus, but she had plenty of other reasons for the continued fever, chills and generally sick appearance in the large number of postules on the back, the abscesses and the enormous carbuncle on her shoulder.

Case 2: A. B., female, age 8 years, was admitted to Dr. George Fetterolf's service at the University of Pennsylvania Hospital on Dec. 29, 1927, with a purulent discharge from the ear, headache and fever.

The history of the present illness showed that the patient had a right earache following an attack of mumps in November. On Dec. 23, she again developed a severe right earache with fever, and on the following morning the drum was incised, with the discharge of much pus. The earache continued, with headache, fever and purulent discharge, until Dec. 27, when the symptoms were relieved, but today she again developed severe pain, headache and fever and was sent to the hospital. She vomited today for the first time.

The family history was negative and the previous medical history was unimportant.

The physical examination on admission showed a fairly well nourished child complaining of pain in the right ear and side of head and rather drowsy. The eyes, nose and throat were negative.

There was a small amount of purulent discharge from the right ear, no bulging of the posterosuperior wall of the canal, and very slight, if any, tenderness over the tip of the mastoid. There was some swelling of the right submaxillary and posterior cervical lymph nodes and some rigidity of the neck. The lungs and heart were negative except for a systolic murmur at the apex, transmitted to the axilla and heard on both sides of the sternum. The extremities were negative and the reflexes normal.

The temperature on admission was 103° F., and after the patient had returned from Roentgen ray examination she developed a chilly sensation. The Roentgen ray films showed clouding of all the cells on the right side, with no evidence of abscess formation. The left side was slightly hazy. A diagnosis of chronic mastoiditis was given. The blood count was as follows: 71 per cent hemoglobin, 22,000 leukocytes, with 91 per cent neutrophiles, 7 per cent lymphocytes, and 2 per cent large mononuclears and eosinophiles. A blood culture was taken.

Although the results of the examination of the ear and the Roentgen ray studies showed a doubtful mastoid involvement, a simple mastoid operation was done on the day she was admitted because of the generally sick appearance of the child. No pus was found until the antrum was reached. The mastoid cells were not broken down but were intensely congested and there was considerable bleeding from the bony structure throughout the operation. All the mastoid cells were exenterated. The plate overlying the sigmoid sinus was exposed, and was of a normal appearance, with no softening of the plate. The sinus was not uncovered. The wound was packed with iodoform gauze and left open.

Following the operation, the temperature went to 105° F., but next morning had gone down to 103° F. Leukocyte count was 13,000.

On Dec. 31, two days after the operation, the temperature again rose to 105° F. and there was noted slight rigidity of the neck and a positive Kernig's sign. Lumbar puncture was made and 15 c.c. of cloudy fluid withdrawn, which showed a cell count of 1,080 with 74 per cent polynuclear leukocytes. Blood count showed a leukocytosis of 18,000, and the urine was practically negative. Blood pressure was 100 systolic. Another blood culture was taken and a culture of the spinal fluid was made.

On the following day two lumbar punctures were made, removing 20 c.c. and 30 c.c. of spinal fluid, respectively. Examination of the fluid showed a cell count of 450. A blood transfusion of 300 c.c. was given from the father. Reports of the blood cultures were negative. Leukocyte count today was 13,400. Next day the high temperature was 105° F., and the low temperature 98.8° F. Cell count of the spinal fluid was 130 cells. Dr. W. J. Gardner was asked to make a Tobey-Queckenstedt test. This test gave on left jugular compression an average rise in five trials of 14 c.m. of water, and on right jugular compression an average rise in six trials of 3 c.m. of water. Dr. Gardner reported that the test showed conclusively a venous obstruction somewhere between the torcular Herophili and the internal jugular vein on the right side.

In view of such a positive diagnosis it was decided to expose the sigmoid sinus. On Jan. 2, this was done after Dr. Ferguson had first ligated the right jugular vein. The sigmoid sinus was uncovered from the knee downward for about an inch. The sinus appeared of the normal bluish color and showed no granulations about it. Pulsation could be felt and the sinus did not have the feel of an obliterating thrombus. After compression of the sinus above and below, an incision half-an-inch long was made lengthwise in the sinus wall, exposing within the lumen a small clot, which was easily picked out with a pair of forceps. On relaxation of the pressure on the vessel there was free bleeding from both ends. A culture was taken from the place in the sinus where the clot was lodged, and also one of the pus in the external canal. Report on these cultures three days later showed a pure growth of hemolytic streptococcus.

On the next day there was a noticeable improvement in the mental condition. The temperature ranged from 102.4° to 98.6° F. Leukocyte count was 14,000. Examination of the eye grounds showed no pathologic lesions, and neurological examination showed no abnormal pathological reflexes and no evidence of brain abscess. A blood

culture was taken, which next day showed on the plate 250 colonies of a pure culture of hemolytic streptococcus. Blood transfusion of 150 c.c. from the father was given, followed by a chill lasting one hour.

During the next few days the general condition of the patient steadily improved. The excursions of temperature were less and the mental condition was much improved. Blood transfusions were given every second day. All cultures of the spinal fluid have remained negative. Blood culture taken on the fifth day showed 26 colonies of streptococcus hemolyticus. The leukocyte count on the fifth day was 16,700, and for the next three weeks the leukocyte count varied between 14,000 and 21,000.

A note on Jan. 13, 15 days after operation, reads as follows: "Patient continues to run an intermittent septic temperature. Blood culture taken yesterday is reported negative. There is no evidence of local metastatic lesions. Heart, lungs and kidneys physiological."

A note three days later says: "Patient is still running intermittent temperature and for the last three days it has been above 102° F. Today she has tenderness, swelling and limitation of motion in the left shoulder region. Roentgen ray examination showed no bone or joint disease of the shoulder."

Two days later, Dr. Ferguson opened a large subpectoral abscess extending as far as the coracoid process, but not involving the shoulder joint. Culture from the abscess later showed pure strain of streptococcus hemolyticus.

From Jan. 18 to 25, the patient continued to run a temperature, varying from about 98.6° F. in the morning to about 102.5° F. in the evening. Repeated medical and urine examinations showed those organs to be functioning satisfactorily, and a neurological consultation gave negative findings.

On Jan. 25, four weeks after the operation, a large abscess of the left labium majus was incised and drained. Culture showed pure growth of streptococcus hemolyticus.

From this time on, the temperature remained practically normal, the child gained steadily in weight and strength, and the abscess cavities and mastoid wound gradually closed. She was discharged on Feb. 5, after a stay of five weeks in the hospital. On the day of her discharge a small abscess was discovered in the left gluteal fold, which was opened next day in the surgical dispensary.

I have seen this patient many times since her discharge. She has not developed any other abscesses, all of her wounds have entirely

closed, she has gained many pounds in weight and is in excellent health.

Comment: This patient presented a condition that I think we can put in the class of hemorrhagic mastoiditis accompanied by a thrombosis at first of the small venules of the mucosa lining the mastoid cells, soon extending to the small veins of the intercellular bony framework and later reaching the lateral sinus through one or more of the small emissary veins. This process of extension has been well described by Kopetsky[6]. When the thrombotic process reaches the veins of the intercellular bony tissue there is a septicemia established with the development of the symptoms of lateral sinus thrombosis, although the sinus has not, as yet, become involved.

In this patient the examination and X-ray report showed a condition that was typical of a hemorrhagic mastoiditis. There was a scanty purulent discharge from the ear, no bulging of the canal wall and very little tenderness of the mastoid, while the X-ray showed only a clouding of the process, without a breaking down of the cell walls. This clouding is due to the intense congestion of the mucous membrane lining the cells, which results partly from the irritation of the infecting organism and partly from the stasis of blood in the venules due to the thrombosis of many of them. Such a condition as this was shown at the time of operation by the excessive bleeding from the mastoid interior, the absence of pus and the presence of intact cell walls.

Another point of interest in this patient was the severity of her illness, which seemed much greater than would be expected from a simple mastoiditis following middle ear infection. She was admitted with a temperature of 103° F., which went to 105° F. that night. She was at times in a delirious state and was very drowsy. She may have had even at that time a septicemia, although the blood culture was negative, but it seems more likely that she had developed the meningitis which later was discovered. This meningitis was apparently only of the serous type, as all spinal fluid cultures were negative.

Another interesting feature of this case was the development of metastatic abscesses. I consider it very fortunate for the patient that these abscesses formed in such areas as the subpectoral region, the labium majus, and the buttocks, rather than in the lungs, the liver or the brain. The distribution of these lesions apparently shows the selective affinity of different types of organisms for different types of tissue. According to Kopetsky[10] it has been found that the pneumococcus and the streptococcus mucosus capsulatus invariably affect the endocranium after invading the mastoid; streptococcus hemo-

lyticus show several strains, one selecting the joints and serous membranes, another the superficial structures, such as the skin and muscles, while still another attacks the lungs.

The findings of the Tobey-Queckenstedt test in this particular case were not entirely substantiated at the time of operation. An obliterating thrombus was not found, but only a small one of the mural type, which could not possibly have shut off the circulation through the sigmoid sinus.

This patient owes much to Dr. Samuel A. Shellburne, the interne on service at that time, whose tireless efforts on her behalf had a great deal to do with her recovery.

BIBLIOGRAPHY.

1. Fraser, J. S.: Septic Otitic Thrombosis of the Cranial Blood Sinuses and Jugular Bulb. *Jour. Laryn. and Otol.*, 39:253, May, 1924.

2. Atkins: A Report of Forty Cases of Lateral Sinus Thrombosis. *N. Y. State Jour of Med.*, 24:884, Oct., 1924.

3. Campbell, Don: Lateral Sinus Thrombosis. The Laryngoscope, 32:775.

4. Rugg, K.: Beitrag zur Frage über Häufigkeit und Vorkommen otitischer Hirnkomplikationen. Ztschr. f. Tals-Nasen-u-Ohren., 19:345, Dec. 27, 1927.

5. White, J. W.: A Study of Lateral Sinus Thrombosis, with Report of Cases. The Laryngoscope, 36:96, Feb., 1926.

6. Kopetsky, S. J.: Intracranial Complications of Suppurative Otitis Media. *J. A. M. A.*, 83:336, Vol. 2, Aug. 2, 1924.

7. Naftzger, J. B.: Thrombosis of the Lateral Sinus. Ann. Otol., Rhin. and Laryn., 33:1297, Dec., 1924.

8. Lillie, H. I.: Infection of the Sigmoid and Lateral Sinus. Surg. Gyn. and Obst., 35:418, Vol. 2, 1922.

9. Yerger, C. F.: Intracranial Complications of Suppurative Otitis Media. *Ill. Med. Jour.*, 46:260, 1924.

10. Kopetsky, S. J.: Acute and Chronic Suppurative Otitis Media. Arch. Otol., 2:379, Oct., 1925.

2117 Chestnut Street.

AN INTERESTING CASE OF LATERAL SINUS THROMBOSIS.*

DR. HENRY S. WIEDER, Philadelphia.

The following case of thrombosis of the lateral sinuses, following acute mastoiditis, is reported not because of its exceptional features, but because several points arose during the study of the case which proved instructive and worthy of discussion.

J. S., male, age 46 years, shoemaker by occupation, had influenza the latter part of December, 1928. After about a week of illness, he developed a discharge from the left ear, the tympanic membrane having perforated spontaneously. There is some doubt whether or not he had any mastoid tenderness at that time. He recovered from his influenza but the ear was discharging for about three weeks, receiving no special care. It ceased to discharge about eight days previous to coming under observation. Five days after the discharge ceased he developed pain over the mastoid, with some fever.

Jan. 26, 1929, three days after developing pain, he consulted an aurist, who, after having an X-ray taken, advised immediate operation, which was refused. The writer saw the patient the same night, finding his temperature about 100°, some tenderness over the lower portion of the mastoid bone, and the tympanic membrane showing some bulging, with no discharge present. A wide incision of the tympanic membrane was made, the patient given the usual treatment of local heat, carbolized glycerin, mercurial purge, etc., and told he would be seen the next morning. As the patient spent a restless night, he was immediately sent to the Northern Liberties Hospital for study and probable operation. His temperature was 99.2° on admission; leukocytes, 10,000; with 87 per cent polymorphonuclear. The mastoid films were obtained and showed some cellular destruction over the posterior portion of the mastoid. As the patient continued to have pain and his temperature went up to 101⅘° the first night in the hospital, operation was urged the following day, but flatly refused. The second day in the hospital the temperature went up to 103⅗°. As the patient continued to have pain, the temperature fluctuating from 99.3° to 102°, he consented to mastoidectomy on the third day in the hospital, Jan. 29. Leukocytes went down to 9,000, with 85 per cent polys on the day of operation. Urine nega-

*Read before the Philadelphia Laryngological Society, April 2, 1929.

Editor's Note: This ms. received in The Laryngoscope office and accepted for publication April 24, 1929.

tive. The operation was done under gas anesthesia. Sulcus incision made and on raising the cortex there was immediate discharge of pus from the center of the bone. Probing revealed that it came from beneath the inner plate, being a perisinus abscess. The entire mastoid was necrotic and the dura was exposed for an area over an inch in diameter. The dura was covered with grayish granulations. As far as could be determined, the sinus was normal. The antrum was deep and there was no pus exuding out of the middle ear. Iodoform gauze was introduced, and the wound sutured above and below. A plug was introduced in the external auditory canal. Temperature, while coming down to normal immediately after the operation, promptly rose the following day to 102⅖° and thereafter ran a fluctuating course between normal and the above figure, but showing a gradual downward tendency, so that on Feb. 2, the highest point reached was 99⅖°. As patient complained of being slightly chilly, had some pain, his temperature showing an upward trend, leukocytosis had gone up to 15,400, with 85 per cent polys, he was given 20 c.c. of Pregl's iodin daily for two days. While the temperature showed an intermission on one occasion to normal, it nevertheless rose again, so that on the second month, the sixth, a spinal puncture was performed, removing 3 c.c. fluid for examination and then performing a Toby-Ayer test, using a mercury manometer. The spinal fluid pressure was 8 m.m. of mercury. Making pressure over the left jugular, the diseased side, the pressure slowly rose to 15 m.m. of mercury and then receded on release of pressure to 8 m.m. On making pressure over the right jugular, the healthy side, the mercury rapidly rose to 35 m.m., and as rapidly receded on release of pressure. The experiment was repeated on each side, with exactly similar results, so that immediate opening of the lateral sinus with tying of the jugular vein was advised, but again absolutely refused. Examination of spinal fluid withdrawn revealed a cell count of 20 leukocytes, albumin +, sugar +. The blood culture taken on Feb. 6 remained negative for four or five days and then showed some contamination with bacillus subtilis, which is the common laboratory contaminant, but also some small cocci, the nature of which were not determined, due to sudden illness of the technician.

As an alternative effort to stem the tide of infection, 15 c.c. of 1 per cent mercurochrome was given intravenously, which was followed by a chill and a rise of temperature to 103⅖°, and a fall to 94⅘°. Temperature remained normal the entire next day and the leukocytes were 13,600, with 83 per cent polys.

As the patient's temperature rose that night to 103⅕° and he had another chill, he finally consented to operation, which was performed

at 8 o'clock the next morning. Before attacking the mastoid wound again, the left jugular vein was exposed, tied between two ligatures, and severed. A rubber dam drain was inserted and the wound sutured and dressed. The mastoid wound was then reopened, granulations curetted away, and the sinus was exposed more thoroughly by removal of a considerable amount of bone and then freely incised, allowing the escape of reddish, liquefied pus and softened blood clot. There was no bleeding from either end of the sinus. More bone was removed and the sinus opened widely and a small curette introduced about 1½ in. toward the torcula and dark red, solid clots removed, with still no bleeding from the sinus. Curette was introduced down to the jugular bulb and clots removed without any bleeding. As it was not thought advisable to do anything further, the ends of the sinuses were packed with narrow-strip iodoform gauze and the wound partially sutured. The culture of the pus obtained from the lateral sinus showed staphylococci with a few pneumococci.

Following the second operation, the patient's temperature rapidly showed a downward course, reaching practically normal the fourth day after operation. The urine output was rather low, despite the forced intake of fluids, probably due to the inhibition by the mercurochrome. However, it never was alarming. The general condition progressed favorably with the exception of persistent headache at nights, rarely in the daytime. The leukocyte count remained about the same, but the polymorphonuclear rose, reaching 89 per cent four days following the operation, with the leukocyte count 12,400. As the headache and high polys count created a suspicion that we might still be dealing with further intracranial complications, the patient was carefully studied from the standpoint of abscess of the brain. He was seen in consultation with Dr. Yaskin, who gave a negative report. He also had his eyes examined by Dr. Herman, who found nothing pathological. Cold douching of the right or normal ear showed the horizontal and vertical canals active, causing both nystagmus, past pointing and violent vomiting. Douching the left ear with cold bichlorid solution failed to give any nystagmus or past pointing but produced violent vomiting, practically ruling out posterior fossa involvement. He was gradually gotten out of bed and sent home from the hospital on Feb. 23, 29 days after admission to the hospital. His wound continued to fill in and became practically dry for about two weeks, and then it suddenly started to discharge again on March 18. There is a small sinus about ½-in. deep, but there are no evidences of exposed bone and the cause for the sudden

discharge cannot be stated. The patient is perfectly well, doing his daily work with just a minute sinus behind his ear.

The points of particular interest to the writer have been the following: *1.* The question whether it is necessary or wise to go back to or beyond the torcula in cases with extensive thrombosis. *2.* The fact that the Toby-Ayer test, while extremely valuable, cannot be accepted as demonstrating a mural clot as opposed to an obstructing clot. *3.* The cause for the persistence of headache after removal of the focus infection. *4.* The persistence of the high polymorphonuclear count after operation.

Considering these points seriatim, the reason the writer did not feel justified in pursuing the clot further was the knowledge of the fact that thrombi are usually aseptic in the beginning and become infected later in the progress of the case, and that the distal portions of the thrombus are usually sterile until late in the disease. In this case there was such a marked difference in appearance of the softened thrombus to the mastoid and the firm red appearance of the distal portions of the thrombus that he considered it was safer, with good drainage, to stop short of the torcula rather than to take a chance of causing infection in the other sinuses by breaking down barriers and introducing infected instruments into the clean area.

With reference to the Toby-Ayer test, while this proved absolutely diagnostic and theoretically should be able to differentiate between a mural and an obstructing clot, nevertheless, the fact remains that this case was most evidently obstructive long before the test was made. Nevertheless, there was a rise of spinal fluid pressure of 7 m.m. of mercury, twice in succession when pressure was made over the affected side. It has been claimed this is due to tenseness of the patient, holding the breath, or some other extraneous cause; even if this is the case, the writer considers it impossible to eliminate such factors unless the patient is under an anesthetic at the time the test is made. Probably the correct explanation of the phenomenon is, however, that a slow rise is obtained when the inferior petrosal sinus and the jugular bulb on the affected side are not thrombosed but no rise is obtained when either of these sinuses are obstructed. Therefore, too much stress should not be placed upon this point of rise in pressure as indicating a mural thrombus as against an obstructing thrombus.

With reference to the persistent headache, Eagleton has pointed out the violent changes that can occur in the brain as the result of cutting off the circulation in the jugular. This is probably the explanation of the continuance of headache. The persisting high poly-

morphonuclear count does not lend itself to ready explanation, but in the absence of ability to find any other focus, it will have to be taken in the light of a reparative phenomenon, trusting that it may not still be an evidence of some latent focus.

Finally, the thought arises, *especially in cases where there is some possible suspicion at the primary operation, of a possible thrombosis being present, and particularly in those cases of perisinus abscess where the condition of the sinus is masked by the presence of granulations, whether it would not be good practice to perform a Toby-Ayer test at the time of operation* in preference to leaving the matter in doubt, or making a needle puncture, which may carry infection into a normal sinus or give inaccurate information. While there is the merest possibility of danger in the Toby-Ayer test of dislodging a loose thrombus by the sudden rise of intravenous pressure, this is only theoretical as far as my knowledge goes, and the danger is far outweighed by the information gained. The only other objection to the Toby-Ayer test has been the possibility of infecting the spinal fluid by a withdrawal of any of it in the presence of a bloodstream infection. It should not, however, be necessary to withdraw enough fluid in performing this test to subject the patient to any danger whatever. In the opinion of the writer, *the chief precaution to be taken in performing this test is to be sure that the connecting tubes are long enough and sterile so as to take care of any fluid that may leave the canal when the sudden high rise occurs following pressure over the healthy side, with just as sudden fall and return of fluid to the spinal canal on release of pressure.*

2106 Spruce Street.

A CASE OF RECOVERY FROM AN OTOGENOUS BRAIN ABSCESS AFTER RUPTURE INTO THE LATERAL CEREBRAL VENTRICLE.

Dr. Arthur Kovacs, Milwaukee.

Recovery from an otogenous brain abscess with rupture into a ventricle of the brain is an exceptional event. Otto Koerner in his classic monograph cites only one case, that of Goerdt[1], and W. Uffenorde has published since another case[2]. The rupture is the terminal stage of the brain abscess, which, together with the communication into the arachnoid cavity, the progressive encephalitis and brain edema, is the cause of death.

A nonfatal case which came under my observation while in an already advanced stage ranks among such rare and fortunate exceptions. The patient is, after 2½ years, without subjective symptoms, thus confirming the rather recently accepted dictum that endocranial complications, even in very advanced and seemingly hopeless state, do not contraindicate operation, since there is a definite possibility of recovery.

The essentials for a fortunate result are a very early and correct diagnosis, operation at the right time and proper after-treatment. In the case to be described it was impossible to satisfy all of these prerequisites, because the patient was not under medical care in the prodromal and latent stages of his brain abscess, it being necessary to operate within three hours after first seeing him, thus rendering a proper examination impracticable and correct diagnosis difficult.

As it was a case of a right temporal brain abscess the focal symptoms were lacking, which, had they been present, could only have been interpreted with difficulty in view of the grave condition of the patient. Of the three symptom groups, *i. e.*, general constitutional symptoms, general brain symptoms and local brain symptoms, only the first two groups were useful in differentiating a meningitis from a brain abscess or confirming the coexistence of both conditions.

Recognition of the presence of an endocranial condition and its otogenous origin was not difficult in this case, because the otherwise deficient anamnesis was quite complete in this respect, and the objective ear findings easily betrayed the chronic cholesteatomatous ear

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication April 25, 1929.

process and its state of acuteness. The endocranial complications indicated the necessity of an urgent operation.

The most conspicuous elements of the history are as follows: E. B., a young man of 18 years of age, was taken to the Hanover Hospital, Aug. 21, 1926. The patient's mother related that he had had whooping cough, chickenpox and measles during childhood, and that following an attack of coryza the right ear had started to run. Since then it had discharged at various times but had never received medical attention. About two weeks previous to admission to the hospital it started to discharge again, accompanied by a headache on the right side, which gradually became more severe. He suffered repeated attacks of vomiting, became bedridden, refused to eat, slept constantly, at times showed delirium, and for the past 24 hours had not responded to questions.

The physical examination showed that the patient was very emaciated, face grayish in color, lethargic. He opened his eyes to loud questions, grumbled indefinite words, reacted slowly to external stimuli. There was pronounced rigidity of the neck, positive Kernig, sluggish pupillary reactions; test of hearing, caloric, turning and fistula experiments impossible. No spontaneous nystagmus. Patellar reflex present; no opisthotonous; facial nerve unaffected; tests for sensation impossible. Fundus of the eye negative. The ear examination showed an external meatus of normal width. In the fundus and the middle ear was observed a very penetrating, greenish-yellow pus, mixed with cholesteatomatous masses. Entire destruction of the drumhead, destruction of the ossicles, the attic was open and showed a necrotic lateral wall. The skin over the mastoid process was normal in contour and color, pressure or percussion producing no reaction on the part of the stuporous patient. Temperature, 103.2° F.; pulse, 62.

The operation was performed three hours after admission. The corticalis of the mastoid process was hard, the lower part sclerotic, while progressing toward the antrum was found a bone abscess about the size of a bean, showing a carious wall and connected with the antrum, and with various small abscesses originating from the conglommerate of mastoid cells. The sinus was free, the upper third of the descending portion being covered with granulation tissue. Behind the tegmen tympani was free dura about the size of a penny with dirty greenish-yellow discolorations of purulent softening. Upon slight pressure about half an ounce of thin, greenish, not very fetid pus oozed out from the brain substance through a fistula communicating with the temporal lobe. Bacteriological examination revealed

streptococci. On account of the irregular pulse and embarrassed breathing, operation was discontinued. Loose iodoform gauze was packed into the wound.

Upon lumbar puncture a slightly turbid, sterile fluid was obtained.

The operation was not followed by any improvement in the condition of the patient. He continued stuporous, occasionally moaning and uttering senseless words. The temperature rose to 103° and the pulse to 96.

Aug. 22: The day following the operation the temperature had not subsided, being intermittent in type and accompanied by bradycardia. The stuporous condition alternated with epochs of agitation, jactitation, efforts to jump from the bed and to tear off the dressings, and at times he arose and walked around the ward. He paid no attention to efforts to quiet him, being unable to answer, and did not recognize his surroundings. Daily change of dressings, the gauze drains being found soaked with pus.

Aug. 24: Bandage changed. Temperature improved, 99.2°; slept more quietly; able to answer questions with effort. Positive Kernig, cranial nerves free, nystagmus negative, heard conversation at concham; eye fundus normal.

Aug. 25: Temperature higher, 99° to 101°; pulse, 80 to 88. Noisy, restless at night; delusions that strange people were chasing him.

Aug. 26 to 28: Condition practically unchanged. Temperature, 102.6°; pulse, 70 to 92. Urinary incontinence. Daily change of dressings showed wound to be healing satisfactorily, the necrotic areas loosening and granulation tissue filling in the cavity. The dura mater showed no change. Decreasing amount of pus on the drain, the odor of which was less fetid. Stretching of the wound with the Killian nose speculum was omitted, as well as palpating with the finger to determine the presence of an abscess membrane.

Aug. 29: Condition very poor, on account of which an exploration of the wound was done. Upon exposing the dura around the already free portion, normal color and consistency were observed. Trial puncture in different directions revealed no pus. Chiselling away the posterior wall of the external auditory canal to the horizontal semicircular canal showed fetid cholesteatomatous masses and pus in the middle ear. Tegmen tympani normal.

Aug. 30: The stuporous condition was followed by restlessness. Temperature, 99.2° to 100°; pulse, 60 to 64. On changing the bandage, the bradycardia subsided rapidly, the pulse rising from 64 to 94, being weak, irregular and at times dropping beats.

Aug. 31: Shivering; temperature, 99.6°; pulse down to 64. Changing the dressing, the pulse rose to 60 and became fuller.

Sept. 1 to 4: The same critical condition, the shivering repeating itself. Temperature, 99° to 102.6°; pulse down to 37. At times painful moaning, took little nourishment.

Sept. 5: On changing the dressing, a serious turn for the worse (generally considered as a lethal end of cerebral abscesses). After pulling out the gauze drain, clear serum pulsated from the abscess cavity, overflowing the edges of the wound and being stopped by immediate closing of the abscess cavity with a gauze tampon. The quantity of fluid was 1 to 1½ ounces, there being marked cerebrospinal pressure, as one observes during a spinal puncture. A deep stuporous state followed but the weak, irregular pulse rose from 34 to 66, becoming stronger and more regular.

Sept. 6: Loosening the gauze packing produced the same phenomenon as on the previous day, but showed less pressure. Bacteriologic examination revealed streptococci, possibly a contamination of the fluid as it flowed through the wound cavity.

Sept. 7 to 8: Gradually decreasing discharge of fluid. Condition of the patient critical, deep coma alternating with terrifying delirium several times daily. Temperature, 100° to 103.8°; pronounced bradycardia; no signs of paralysis of the cranial nerves or paresis of the muscles of the extremities. Urinary incontinence still present. There was a conspicuous contrast in the patient's general condition with that of the wound cavity with its fresh, healthy, rapidly-growing granulation tissue.

Sept. 12: Striking improvement in the sensorium; patient recognizes people; mutters a few intelligible words. Temperature, 99.6° to 100°; pulse, 70 to 72 . During the following days the wound healed rapidly, the abscess cavity filling in, and only traces of pus showed upon the superficially-placed drains.

Sept. 16: Abscess closed. Marked improvement in the general condition of the patient. While fairly good and quiet during the day, at night epochs of excitation almost reached delirium, especially in Sept. 19, 20 and 21. Cried frequently, thought strangers were pursuing him, had visual hallucinations (saw children around him); jumped from the bed, ran into the adjoining rooms and walked upon the beds.

Sept. 25: Vomited once. Complained of headache. Temperature. 98.4° to 100°; pulse, 76 to 86. Temperature gradually decreasing.

Sept. 27: Vomited during the morning hours. Temperature normal. General condition and strength rapidly improving.

Oct. 1: Patient left the hospital.

The patient did not report for observation again until May, 1927, at which time he appeared in excellent general condition, without the slightest subjective symptoms. The wound behind the ear had closed, with exception of a depression the size of a pea. The external auditory canal was narrow, the cavity of the radical operation well preserved, and only a moderate amount of pus in the middle ear.

On Aug. 1, 1927, a Panse plastic operation with smoothening up of the bone edges and curettage of the tuba ostium and its surroundings was performed. In eight weeks the wound was thoroughly epithelialized, being smooth, dry and glazed.

Otitic brain abscess, associated with chronic otitis, is more frequent than that with acute inflammation, but the prognosis is worse. The statistics of experienced authors differ considerably, but the average mortality is between 59 and 76 per cent. It is much higher in cases of cerebellar abscess, O. Beck's published series showing a mortality of 100 per cent in 41 cases. The cause of death is usually an encephalitis with a leptomeningitis.

The cholesteatomatous ear process frequently described in the literature was also present in this temporal lobe abscess, coming after the primary bone disease and localizing adjacent to it on the bony top of the mastoid process. During the operation a fistula was found between the abscess and the cavity of the mastoid process, making it very probable that a part of the contents of the abscess had previously discharged through the natural ear canal.

While there were no symptoms or macroscopic pathological changes in the sinus, it is very difficult to state the extension of the inflammation along the leptomeninges. We may presume a serous leptomeningitis from presence of the marked Kernig sign, which is a special meningeal symptom.

The presence of an encephalitis with progressive extension after the operation was proved by rupture of the cavity into the lateral ventricle five days after the operation, by the long duration of the psychical derangements, and by the conspicuous bradycardia. Evidently long before operation there were adhesions between the pia and the surface of the brain which prevented the development of a diffuse meningitis. The presence of a serous meningitis was proved by the increased pressure of the cerebrospinal fluid.

The absence of pathological changes in the eye fundus is not uncommon, according to the experiences of various authors. O. and E. Ruttin always found the fundus normal in cases of temporosphenoidal abscesses, while some even consider an optic neuritis or choked disc as evidences against an otogenous abscess.

It was very interesting to note the cessation of the bradycardia immediately after the changing of the bandages. No symptoms suggesting pressure on the internal capsule were noticed, there being neither irritation nor paralysis of the motor paths, which could be expected in view of the large size of the abscess.

The condition described by Foster Kennedy as "temporosphenoidal epilepsy" and stated to be a conspicuous sign in temporal abscesses was also present in this case. The patient remaining stuporous for hours, became restless, especially at night, and was tortured by visions and hallucinations.

Of the three lateral ventricles (posterior, inferior, lateral), it was through the inferior that the abscess ruptured, as it is the only one which extends to the temporal lobe.

Very evidently, the inflammation on the surface of the ependyma of the lateral ventricle raised the interior pressure and produced the rupture, and the same factor prevented the regurgitation of the abscess contents with production of a fatal generalized meningitis.

The question might arise as to whether the liquid exuding from the abscess was of cerebrospinal origin. The abscess was kept open by loose gauze strips so that retention of a considerable amount of liquid was impossible. The large quantity of clear serum could not have originated from an abscess cavity, nor would it have pulsated upon being liberated (a sign of marked pressure). The flow of liquor repeated itself on four successive days, which would have been impossible had it obtained from the abscess itself. The source of the fluid could be traced directly into the ventricles and the central canal which connected with the abscess cavity through an open fistula.

As far as the writer knows this is the third case of otogenous brain abscess with recovery to be reported, and for that reason he deems it worthy of being recorded in the literature.

BIBLIOGRAPHY.

1. Arch. fur Ohrenheilkunde, 98.

2. Zeitschrift fur Hals-Nasen-und-Ohrenheilkunde, XVIII.

123 Wisconsin Avenue.

TUBERCULOSIS OF THE TRACHEA.*

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The anatomical position of the trachea, which forms, as it were, a connecting link between the larynx above and the bronchi below, would seem to predispose it to frequent tuberculous affections.

And indeed, at first sight it is difficult to explain the comparatively rare occurrence of tuberculosis of the trachea, entering as does the latter into close contact with the highly infectious products of tuberculous ulcers of the larynx and in no less close connection with the injurious products of disintegration of tuberculous pulmones. If in addition to this, attention be called to the fact that the biological immunity of the body becomes very low at the period of developed disintegration of tissues affected by tuberculosis, a specific capacity of the trachea to resist such powerful infectious agents, as supplied by tuberculous ulcer surfaces is most strikingly evidenced.

The difficulty of diagnosing tracheal diseases in general and tracheitis in particular may possibly account for their frequently escaping correct identification, with the obvious exception of cases dealt with on the dissecting table. Considerable diversity of published opinion as to the frequency of tracheitis tuberculosa may be likewise explained by the heterogeneous source of the corresponding statistical material, whether clinical or anatomical (such as recorded from the dissecting room).

Thus, Prof. Voyachek[1] holds that tuberculosis of the trachea appears in the terminal stage; Heinze (quoted by Schneider[2]) found 8 per cent of tracheal tuberculosis in 1,226 autopsies; Willigk, 3.6 per cent; Dobromilsky[3], 25.2 per cent of tuberculous changes in the trachea; Loui (cited by Golm[4] failed to find a single case of tuberculous changes in the trachea; Peloso[5] in all cases of pulmonary tuberculosis noted changes in the upper air passages without further specification. With such divergent evidence on the frequency of tracheal tuberculosis, it is obvious that most reliance should be attached to such figures only which have been obtained from numerous autopsies, but even then much discrepancy is manifested by the authors quoted, as displayed by the 8 per cent of Heinze, 3.6 per cent of Willigk, and 25.2 per cent of Dobromilsky.

From the materials collected by us during the period from January, 1925, to January, 1929, at the Pathologico-Anatomical Section

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Editor's Note: This ms. received in The Laryngoscope office and accepted for publication July 29, 1929.

of Mechnikov's Hospital in Leningrad, it appears that in the dissecting room of that section, 2,584 corpses exhibited organs affected by tuberculosis, including also cases in which death was not caused by tuberculosis (but,for example, by cancer of the esophagus). In every one of the above-mentioned cases of autopsy, however, the presence of more or less evident symptoms of tuberculous affection was established (such as chronic fibrous apicitis pulmonum). Within the same period a postmortem examination of 4,328 corpses was made, of which 2,379 cases proved tuberculosis to be the fundamental disease.

In the annexed table the cases of pulmonary tuberculosis collected by us are subdivided according to the following forms of that disease.

TABLE I.

Age	Preem. productive forms				Preem. exud. forms				Mixed character of changes				Acute miliary tuberculosis			
	With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Under 20	45	54	15	7	15	17	5	4	10	6	8	14	....	1	3	5
Fr. 20 to 30	272	196	34	21	59	53	14	4	58	55	13	7	....	1	5	2
Fr. 30 to 40	250	156	53	17	39	30	3	4	60	38	7	8	1	1	3	1
Fr. 40 to 50	209	72	80	23	14	4	3	2	41	13	13	3	....	....	3	..
Above 50	153	52	111	31	5	9	4	1	30	11	13	8	....	....	1	1
Total Amt.	929	530	293	99	132	113	29	15	199	123	54	40	1	3	15	9

From this table it may be seen that most of the cases recorded are referred to the pre-eminently productive forms with caverns giving 56.5 per cent of all autopsies, then follow productive forms without caverns—15 per cent (of 392 productive forms without caverns—79 may be referred to cases with pre-eminently cirrhotic changes in pulmones); the forms with a mixed character of the changes and with caverns constitute 12 per cent; pre-eminently exudative forms with caverns, 9.9 per cent; exudative without caverns, 2 per cent; mixed forms without caverns, 3.6 per cent; acute miliary tuberculosis, 1 per cent (cases in which autopsies concurrently with a generalized abundant eruption in the lungs and other organs exhibited fully dveloped symptoms of chronic tuberculosis, were not included in this group of acute miliary tuberculosis, but were referred to other groups, in correspondence with the character of their chronic tuberculous changes).

Affection of the larynx was determined in 19 per cent of the total autopsies. It should be noted that in this and other tables all the

returns refer to morbid changes determined by autopsy alone, *i. e.*, macroscopical, such as are usually specified in inquests.

TABLE II.

Laryngeal Tuberculosis Without Simultaneous Tracheal Tuberculosis

Age	Preem. productive forms				Preem. exud. forms				Mixed character of changes				Acute miliary tuberculosis			
	With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Under 20	9	9	....	1	6	3	2	....	2	2	....	....	....	....	....	..
Fr. 20 to 30	65	42	5	1	23	16	3	1	21	9	1	....	....	....	....	1
Fr. 30 to 40	48	28	15	3	10	10	1	....	14	9	....	1	....	....	....	..
Fr. 40 to 50	52	10	5	2	5	....	....	....	13	1	3	....	....	....	....	..
Above 50	17	6	5	2	1	....	....	....	7	2	1	....	....	....	....	..
Total Amt.	191	95	30	9	45	29	6	1	57	23	5	1	....	....	....	1

10 per cent of the total number of autopsies with tuberculous changes. In all 493 cases.

TABLE III.

Tuberculosis of the Larynx and Trachea.

Age	Preem. productive forms				Preem. exud. forms				Mixed character of changes				Acute miliary tuberculosis			
	With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Under 20	2	4	....	....	....	....	....	....	4	2	....	1	....	....	....	..
Fr. 20 to 30	29	17	1	2	5	6	1	....	5	9	....	1	....	....	....	..
Fr. 30 to 40	38	14	2	1	4	1	1	....	11	3	....	....	....	....	....	..
Fr. 40 to 50	21	5	2	....	2	1	....	....	4	1	....	....	1	....	....	..
Above 50	9	4	3	....	....	....	....	....	1	....	1	....	....	....	....	..
Total Amt.	99	44	8	3	11	8	2	....	25	15	1	2	1	....	....	..

In all 219 cases. 8.48 per cent of the total number of autopsies with tuberculous changes.

TABLE IV.

Tuberculosis of the Trachea Without Concurrent Laryngeal Tuberculosis.

Age	Preem. productive forms				Preem. exud. forms				Mixed character of changes							
	With caverns		Without caverns		With caverns		Without caverns		With caverns		Without caverns					
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.				
Under 20	3	2	....	....	....	1	....	....	....	....	....	....				
Fr. 20 to 30	9	6	1	....	1	....	....	....	3	....	2	....				
Fr. 30 to 40	9	5	1	....	1	....	....	....	3	2	....	1				
Fr. 40 to 50	11	2	1	....	....	....	....	....	2	2	1	....				
Above 50	8	....	....	....	....	....	....	....	....	2	....	....				
Total Amt.	40	15	3	....	2	1	....	....	10	4	3	1				

In all 79 cases. 3 per cent of the total number of autopsies with tuberculous changes.

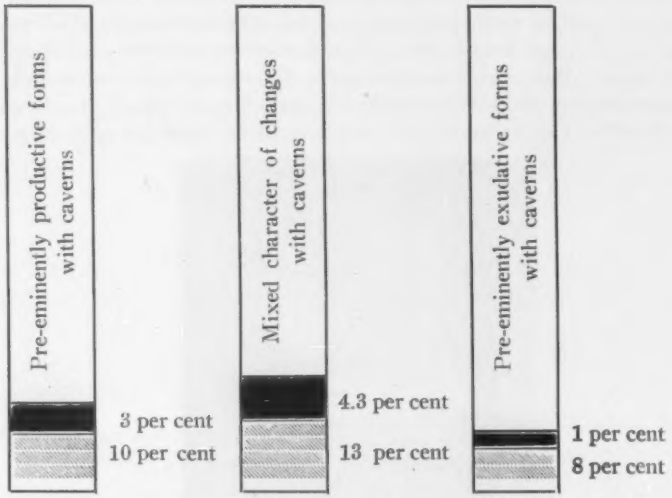
Table V graphically illustrates the frequency of tuberculous affection, both of the trachea and larynx, as also that of the trachea alone. Simultaneous tubrculosis of the larynx and trachea is represented by oblique, of the trachea alone by horizontal shading.

As may be seen from Tables III and IV, the trachea appears to be affected by tuberculosis in 8.48 per cent + 3 per cent = 11.48 per cent of the total number of autopsies exhibiting tuberculous changes.

The most flourishing age, between 20 and 40 years, dominates in our material, as also in that of other authors. As regards the prevalence of either sex among the sick, there are indications in literature to the effect (Muthu[6]) that in Calcutta, for instance, the mortality from tuberculosis of women is six times higher than that of men, which can be partially explained by women spending the larger part of their lives in insanitary conditions. Tadashi Satow[7] emphasizes the fact that in Japan the mortality from tuberculosis of women is always higher than that of men; Holtermann[8] notes the increase of sickness from tuberculosis during war and the period of inflation.

Table III shows that tuberculosis of the larynx concurrently with that of the trachea (in absolute numbers) occurs most often in cases of tuberculosis of the lungs with pre-eminently productive changes and with caverns (this group, as is evident from Table I, is the predominating form in our material) in 143 cases out of 219 cases of tuberculous larynx and trachea, constituting 10 per cent of the total amount of all autopsies with pre-eminently productive changes and with caverns; mixed forms with caverns represent 13 per cent of all autopsies exhibiting tuberculous disease of the larynx and trachea. Eight per cent of all cases of tuberculosis laryngis et tracheae may be referred to pre-eminently exudative forms with caverns. As may be seen from Table IV, tracheal affections in pr-eminently productive cases with caverns, without laryngeal tuberculosis, being anatomically or clinically established, constitute 3 per cent of all autopsies of such forms. In forms with a mixed character of changes and with caverns, tracheal tuberculosis was identified with 4.3 per cent of autopsies of such forms. In examining returns of Table IV and comparing them with those of Table III it appears that for 493 cases of laryngeal tuberculosis (Table II) without a simultaneous affection of the trachea, 219 (Table III) + 79 (Table IV) or = 298 cases in all, may be assigned to cases of such simultaneous affection of the larynx and the trachea or of the trachea alone, or, in round figures, in 500 cases of laryngeal tuberculosis without a simultaneous affection of the trachea, occur 300 cases with such an effection, or about 60 per cent.

Cases of tracheal tuberculosis being thus not so frequent when compared with the general number of autopsies exhibiting tuberculous changes, yet when more carefully examined and compared with cases of laryngeal affections are not of such rare occurrence.



How can the comparatively rare affection of the trachea in comparison with the frequent occurrence of laryngeal tuberculosis be explained? Being situated in a tract easily accessible to infection from two extensive foci, such as are afforded by the diseased lungs, the trachea is liable to be bathed by the products of these affected organs, which settle on its walls, while being also subject to the intrusion of descending streams of injurious matter from the tuberculous ulcers of the larynx, whence it would seem the trachea is destined early to become the seat of a particularly morbid form of tuberculous affection; if to this contact infection of the tracheal walls by the infectious tuberculous discharges be added liability to hematogenic or lymphogenic infection, to which the very anatomical position of the trachea cannot but contribute, it would seem natural to expect the trachea to be subject to the most virulent attacks of tuberculous infection. And if tracheal tuberculosis does not comparatively occur so frequently, this, in agreement with the present state of our knowledge, in our opinion, be possibly due to a local constitutional particularity of this organ. The trachea is generally capable of offering a fairly energetic resistance to injurious agents. Thus, Chodiakoff[9] has

shown that in miliary tuberculosis on the walls of the trachea occur tracts covered with flattened, instead of the normal ciliated epithelium; Maximov (cit. after No. 9) has noted, that by the destruction of the cylindrical ciliated epithelium of the trachea occasionally appears in its place multistratified flattened epithelium. The examination of our microscopic series of trachea affected by tuberculosis has revealed in single cases the substitution of flattened for ciliated epithelium. Such a reaction to injurious agencies by means of the substitution for more highly differentiated elements (such as ciliated epithelium) of lower forms (such as stratified flattened epithelium)

Fig. 1. Throughout the entire trachea occur many small tubercles occasionally—circular ulcers (autopsy No. 1107, 1928).

capable of restoring a continuous tracheal surface—may evidently constitute one of the compensating factors in the struggle for preserving the integrity of deeper-seated tissues, glands and perichondrium, the destruction of which could threaten the normal functions of that organ. The above-mentioned capacity of modifying the epithelial covering of the tracheal walls under the influence of injurious stimulants cannot, however, be regarded as being the only means of protection possessed by that organ. The entire mucous membrane of the trachea is evidently not a suitable object for the development of tuberculous infection and neither does the most usual route for the diffusion of tuberculosis by way of the lymphatic vessels particularly conduce to frequent infection of the trachea by tuberculosis. Yet numer-

ous lymphatic vessels proceeding from the lungs (Zernov[10]), join in the porta pulmonis and ascend by way of the primary bronchi to the glands, below the bifurcation and still higher along the sides of the trachea), which should contribute in a high degree to a rapid development of the infection. The general conditions of the vital activity and the functions of tracheal walls and particularly of their mucous membrane, their as yet undefined chemicobiological features and embryological characters, or, in other words, all that implies *local constitutional conditions*, are predominant in the rise, development and prevention of tuberculous infection.

Fig. 2. Pre-eminently tuberculous affection of larynx and trachea (autopsy No. 1099, 1928).

According to the embryogenetic theory of Schminke (cit after No. 11), which maintains that the development of tuberculosis in any organ is connected with certain features in the development of the embryo, the simultaneous affection of the lungs, larynx (and ergo of the trachea) and the intestines is explained by their common origin from the entoderm. And as a matter of fact we have found in our material frequent cases of intestinal tuberculosis with a simultaneous affection of the larynx (in 287 cases out of 493), of the larynx and trachea (in 166 cases out of 219), and of the trachea without affection of the larynx (in 53 cases out of 79).

The affection of organs, derived from the entoderm and in particular of the trachea—the most resistant of the group: lungs, larynx,

trachea, obviously manifests the complete failure of the recouping powers of the organism, implying a hopeless prognosis. And independent affection of the trachea without a simultaneous attack on the larynx may likewise be explained by the enfeebled constitution of the subject, whence a lethal issue is reached before laryngeal tubrculosis had time to develop (usually a less resistant organ in respect to tuberculosis).

An examination of a number of excised tracheae, affected by tuberculosis, proved the difficulty of combining all the different forms of disease applicable to every individual case.

Fig. 3. Pre-eminently ulcerated affection of trachea.

The classification proposed by Manasse[12] for the tuberculous affections of the larynx, as: *1.* infiltration, *2.* ulceration, *3.* perichondritis, and *4.* tuberculous "geschwülste", does not embrace all the possible forms of that disease; a long time since Prof. Simanovsky[13] observing that the trachea may develop infiltration, ulceration and miliary tuberculosis, he failed, however, to propose a more comprehensive scheme of such tuberculous affections. Recently a number of writers (Voznesensky[14], Dobromilsky[15], Basenko, Ewart Martin[16] and others) attempted the construction of such a comprehensive scheme of the tuberculous affection of the larynx. It cannot, however, be fully applied on account of the numerous and complex symptoms manifested by tuberculosis in this part of the body. We cannot but admit

the truth of the assumption at first advanced by Bumba[17], that the essential part of tuberculous affection consists not only in anatomical changes, but in an uninterrupted struggle between the organism on the one hand and the infective agents on the other—by which these endless variations in the pathologoanatomical features of tracheal tuberculosis may to a certain extent be explained. Permanently changing correlations of the forces of infection with those of the tissues cannot but affect the resultant pathologoanatomic symptoms, whence Bumba's reasonable conclusion as to the somewhat artificied character of the attempts to classify tuberculous affections of the

Fig. 4. Tracheitis tuberculosa with ulcers and tubercles.

larynx (and also evidently of the trachea) seems to be well grounded. If it be borne in mind that a prolonged period of tuberculous toxemia causes the modification of the endocrine system (cit. No. 11), then the profound constitutional changes, due to such a reconstruction of the organism, in altering the resisting capacity of the tissues and their predisposition to infectious agents, afford no basis for a correct determination of the character of the affection, which has not only passed through different stages of the development, but has been accompanied by periods dissimilar from one another in the changes of the organism itself.

In studying forms of tracheal tuberculosis as exemplified in numerous autopsies we wonder at the frequency of mixed forms: thus, simultaneously with all but indistinguishable tubercles, we often

encounter diversified forms of tracheal ulcerations, both quite superficial and insignificant, and also very deep with extensive destruction of the entire wall, spreading very widely and accompanied by vast infiltrates. Judging from the photographs appended to this article and illustrating the severer forms of tracheitis tuberculosa, but two subdivisions of this particular affection seem to us admissible: *1.* pre-eminently ulcerated forms, and *2.* pre-eminently tubercular

Fig. 5. Pre-eminently ulcerated affection of trachea, the ulcers occasionally fusing together (a piece of tissue taken from the left vocal cord for microscopic examination) (autopsy No. 1075, 1928).

forms. By such a division we wish to emphasize that pure forms are not as a rule met with. We failed to corroborate a predominating localization of the affection on the posterior wall of the trachea, as is urged by some authors, because in the cases examined by us the affection usually spreads over extensive surfaces, both of the anterior cartilaginous, and of the posterior membranous part. Vast affections with exposure of the cartilaginous base of the trachea are met with but rarely, not more than in about 4 to 5 per cent of cases of all tracheitis tuberculosa.

The most severe tuberculous affections in most of our cases have been met with in the lower part of the trachea.

Microscopic changes due to tuberculosis will form the subject of a future contribution.

What is the mechanism of the infection of the trachea by tuberculosis? From a consideration of its anatomical position, it may be

inferred with much probability that the trachea is open to all the known modes of infection: by contact and through the blood and lymphatic vessels.

The trachea is so well provided with lymph by means of the lymphatic vessels connected with the lungs, the principal seat of infection (see above reference to Zernov), that these tracts may be held as playing a very considerable part in the communication of tuberculosis to that organ.

Fig. 6. Profound destruction of tracheal walls by tuberculosis, with exposure of cartilage (autopsy No. 1123, 1928).

It now remains to consider the other possible ways of infection of the trachea, namely, that by means of contact and by blood vessels. The mechanism of infection by means of contact requires no explanation (particularly when the integrity of the epithelial surface of the trachea, exhausted by the long struggle with infection, has been impaired). The frequent catarrhal inflammations of the tracheal walls may at least contribute to weaken resisting power.

Nor can the dissemination of tuberculous infection by means of the blood vessels, characteristic of the second stage in the spreading of that disease, according to Ranke's classification, be excluded as a cause of tracheitis tuberculosa. A combination of the hemo- and lymphogenous modes of tuberculous infection, when the local constitutional capacities of resistance are impaired is apparently not an unfrequent means of causing tracheitis tuberculosa.

The highly resistant epithelial surface layer of the trachea is an excellent natural protective medium, which loses these properties only in the case of a catastrophic fall in its resisting powers.

As the struggle of the organism, affected by chronic infection, is often but very gradually displayed, we share the suggestions of some English writers (quoted after No. 18) that in all cases of pulmonary tuberculosis objective and not the subsequently occurring subjective symptoms of an affection of the larynx (ergo, of all air passages in general) should be detected. In view of the frequent combination of tuberculosis of the trachea with that of the larynx and the necessity of the strictest economy in the expenditure of the protective forces evolved by the organism in every case of suspected tracheitis tuberculosa and of corresponding method of treatment, much significance and weight should obviously be assigned to the old, but ever new maxim: *"Primum non nocere"*.

LITERATURE.

1. VOYACHEK, PROF. V.: Diseases of the Ear, Nose and Throat, ch. II, p. 219, 1926 (Russ.).
2. SCHNEIDER, SUB-PROF.: Tuberculosis of the Upper Air Passages. 1927 (Russ.). (*Jour. of Ear, Nose and Throat Diseases*).
3. DOBROMILSKY: Specific Changes in the Trachea During Pulmonary Tuberculosis. (Russ.) *Jour. of Ear, Nose and Throat Diseases*, 1928 No. 11-12.
4. HOLM, FRANZ: On Laryngeal and Tracheal Tuberculosis in Its Relation to the Clinic. 1863 (Russ.).
5. PELOSO, GIOVANNI: Il tisiologo e l'otorinolaringologia. Ztrblatt f. H. N. und O. Kunde, Bd. 12, H. 2, S. 65, 1928.
6. MUTHU, C.: A General Survey of Tuberculosis in India. *Brit. Jour. of Tuberculosis*, Jan., 1927, Vol. XXI, No. 1.
7. SATOW, DR. TADASHI: Prevention of Tuberculosis in Japan. *Brit. Jour. of Tuberculosis*, 1926, Vol. XX, No. 3 ,p. 107.
8. HOLTERMANN, VON F.: Lungen und Kelkopftuberkulose in Preussen, in Ruhrkohlen bezirk usw. Zeiterschrift fur Tuberkulose, Bd. 52, H. 5, 1928, S. 397.
9. CHODIAKOV, M.: On the Loss and Regeneration of Ciliated Epithelium of the Upper Air Passages. Vestnik otolaringologic (Russ.), 1928, No. 3, p. 321.
10. SERNOV, D.: Textbook of the Normal Anatomy of the Human Body. (Russ.), Ch. II, 1925.
11. STERNBERG, PROF.: The Constitution and Clinic of Tuberculosis. Vrach. Gaseta (Russ.), 1927, p. 1395.
12. MANASSE, DR. PAUL: Anatomische Untersuchungen uber die Tuberkulose der oberen Luftwege. Berlin, 1927.
13. SIMANOVSKY: Laryngeal Tuberculosis. 1914 (Russ.).
14. VOSNESENSKY, A.: On the Nomenclature of the Clinical Diagnosis of Tuberculosis. (Russ.), 1925, Russkaja Otolaringologia, 70.
15. DOBROMILSKY, TH.: A Contribution to the Question of the Classification of Laringeal Tuberculosis. Russk. Otolaring., 1928, No. 4, p. 380.
16. *Brit. Jour. of Tuberculosis*, Vol. XXII, No. 1, 1928 (Notices of Books).
17. BUMBA, DR. JOSEF: Die Kehlkopf von Standpunkt der immunobioigischen Forschung von Priv.-Doz. Leipzig, 1925 (ref. Russk. Otolar., 1925, p. 163).
18. Diseases of the Nose, Throat and Ear for Practitioners and Students. Edited by A. Logan Turner, Ltd., 1924, Vol. XVIII, No .3, p. 116.

A NOTE ON THE TREATMENT OF VINCENT'S ANGINA

Dr. Philip Frank, St. Louis.

Without intending to add another to the list of "specifics" in the treatment of Vincent's infection (sodium borate, trichloracetic acid, chromic acid etc.) I merly want to record a line of treatment that I have followed for the past nine or ten years in a series of thirty-seven cases in private practice and probably an equal number in dispensary practice which has given uniformly rapid and unfailing results. All the cases were of course verified by bacteriological examination and the effect of the therapy noted thereby as well.

Bearing in mind that Vincent's is a disease-entity caused by two organisms in symbiosis, the fusiform bacillus and the Vincent spirillum, and that each is susceptible to different agents, my procedure is as follows: After removing the membranous coating from the diseased area in early cases, or the caseous, necrotic debris in advanced cases, the raw surface is swabbed with ordinary tincture of iodine (directed against the fusiform bacillus) followed by thorough swabbing with Fowler's solution (directed against the spirillum). I have found that what is lethal to the fusiform bacillus is quite innocuous to the spirillum and vice versa. My experience is that it is unnecessary to employ strong arsenical preparations, such as salvarsan, against the spirillum for it succumbs readily even to a weak arsenical application. In addition I prescribe Fowler's solution internally, 10 drops three times a day, for five or six days.

In no instance has it been necessary to make more than three or four applications, once each day, to obtain a complete disappearance of the organisms and a cure of the lesion. In most of the early cases but one or two swabbings was all that was required. Some of the advanced cases of four, five and six weeks duration, mistaken either for follicular tonsillitis or diphtheria and treated accordingly with an aggravation of symptoms, showed when they came under my observation involvement of the soft palate or uvula or buccal mucosa in addition to coagulation necrosis of almost half of one or both tonsils, leaving deep excavations, but in no case has this treatment failed to clear up the condition entirely within several days.

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Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication June 26, 1929.

International Digest of Current Otolaryngology.

Editor:
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Grove, in the Archives of Otolaryngology for October, 1929, reports a presumptive case of primary cholesteatoma of the temporal bone occurring in an 18-year-old girl.

After reviewing the literature he concludes that although most of the cholesteatomas of the temporal bone are probably secondary in origin yet primary congenital cholesteatoma of the temporal bone probably does occur and with about as much frequency as cholesteatomas in other portions of the skull. ROSENBERGER.

Brunner und Urbantschitsch, of Vienna, in the Archiv. f. Ohrenh., Jan., 1929, 1-120, present a paper on Hereditary Degenerative Deaf-Mutism. They state that developmental abnormalities have always been considered the chief cause. Recently Neumann, Brunner and Mayer have shown that abnormalities of the cochlea may exist without deaf-mutism even where coupled with otosclerosis.

The study of congenital atresia and cretinism has shown that these conditions may be the cause of deaf-mutism. When we add the inflammatory cases we have three groups in which the middle ear is the chief site of the etiologic factor.

They report a case of atresia of the middle ear which was not diagnosable in life. GUGGENHEIM.

Thompson, of Pueblo, in the Journal A. M. A., Oct. 5, 1929, reports a case of Brain Abscess due to Vincent's Angina following extraction of a tooth under procain. The tooth in question was the upper second molar. A few days later the patient developed a Vincent's infection in the mouth and died 24 hours later. Post-mortem examination showed an abscess in the sphenopalatine fossa extending by way of the middle meningeal vein to the longitudinal sinus. There was an abscess in the right temporal lobe which pre-

sented Vincent's on the smear; there was an abscess in both orbits and a thrombosis of the cavernous sinus which also showed Vincent's on smear. MYERS.

Joel, of Rostock, in the Zeitschrift fur Hals, Nasen u. Ohrenheilkunde, Band XXIII, 3, page 227, reports his experiences with the Muck "Adrenalin Probe Reaction". The reaction is said to be positive or pathological when the adrenalized turbinates, when stroked, show a white line. A normal or negative reaction should show a red line. Joel found that when he obtained a positive reaction it indicated an anomaly of some sort suggesting the existence of some organic derangement. FINEBERG.

Kolisch, in the Zeitschrift fur Laryngologie, 1929, XVIII, reports a new method in the treatment old old-standing, suppurating radical mastoid activities. On the assumption that carbohydrate metabolism is intimately connected with epithelial growth and regeneration, it was attempted to increase the glycogen storage in the liver cells and locally to the epithelial cells with the aid of insulin.

Clinically, this discovery was first used in the treatment of chronic ulcers of the leg. The author used insulin in 51 cases of old-standing, suppurating radical mastoid cavities which were otherwise completely satisfactory (good access to cavity, no sequestrum, no recurrence of cholesteatoma). At first he used daily instillation of 1 c.c. of insulin into the cavity, closing the meatus with cotton wool. After a few days it sufficed to mop our the cavity with an insulin-soaked sponge. This stimulated the healing and complete regeneration occurred in many cases. To prevent recurrences, the patient continues to use two or three drops of insulin solution dropped into the cavity through the external canal about twice a week. In some cases chemically pure glucose was insufflated in addition to the insulin application. This seemed to answer better than the insulin alone.

Kolisch reports very striking results. He obtained absolutely dry cavities in from four to eight weeks in cases which, hitherto, had resisted all other treatment for many years. FINEBERG.

Oppikofer, of Basil, in the Archiv. f. Ohrenh., Jan., 1929, 1-120, presents an article on the Pulsation of Pus in the External Auditory Canal and Extradural Abscess. He states that extradural abscess may cause no typical symptoms and when an acute mastoiditis does not show improvement, under usual treatment, this condition should be kept in mind. Mark has stated that pulsation of pus in the canal after the first week or two of an acute otitis, speaks very strongly

for extradural abscess which is in connection with the middle ear pathology. Oppikofer studied 52 cases of extradural abscess, of which 47 were in the posterior fossa, three in the middle, and in two cases both areas were involved. Of this number, 13 cases were without perforation of the drum membrane, leaving a total of 39 cases for special observation as to pulsation. They were all beyond the third week in duration. In 21 of these cases, pulsation was observed; it was absent in 18. GUGGENHEIM.

Friedlander and Sommer, of Vienna, in the October, 1929, Mschr. f. Ohrenheilk., presented an article on the Therapy of Climacteric Ear Noises. They used Sanabo and Klimasan, which are polyhormone tablets, internally, and warm baths with massage and hot pack baths. With the rational use of these measures, they have been able to produce a lessening in 15 severe cases of tinnitus in which all other remedies had failed. FINEBERG.

It is of interest to note that the Royal Society of Medicine, Section of Otology, at their May 3, 1929, meeting, spent considerable time on the discussion of "Treatment of Chronic Deafness". The following gentlemen took part in the discussion: Mr. Somerville Hastings, Sir James Dundas Grant, Dr. P. Watson-Williams, Mr. Alex. R. Tweedie, Dr. Dan Mackenzie, Mr. H. Norman Barnett, Mr. Harold Kisch, Mr. W. S. Thacker Neville, Mr. E. Watson-Williams, Dr. G. Cathcart and Mr. A. Lowndes Yates. These gentlemen discussed many phases of treatment of chronic deafness. The uses of zinc ionization, diathermy, sodium silicate ionization, ossiculectomy, ultra violet light and injection of collosol iodin were all fully described.

Bonain, in the April, 1929, Ann. des Mal. de L'Oreille, etc., presents a resumé of his report to the French Society in October, 1928, on the "Physiology of Hearing". He first presents an historical summary from Vesalius to the present time and the theory of Helmholtz is fully stated. He next discusses various theories on a physical, clinical and experimental basis and concludes with a new theory of his own. The reader is recommended to look up this rather interesting contribution for further information.

FINEBERG.

Werth, of Heidelberg, in the Z. Hals-Usw. Heilk., 23-4, presents a study of the bacterial flora of the tonsils. He bases his statistical report on 43 cases of enucleated tonsils in adults, and 111 in chil-

dren. Some of his conclusions are that there are no characteristic differences of the flora in cases of chronic tonsillitis, with body ailments, and chronic tonsillitis without body ailments. In 111 cases (children), only 15 were free from pathogenic bacteria. In 43 cases (adults), eight were free from pathogenic bacteria. In 34 cases (children), the same flora was found in the adenoids as in the tonsils. In 43 cases (adults), with chronic tonsillitis, 26 had an isolated swelling of the "angina gland" in the fossa retromandibularis. In calling an organism pathogenic, Werth laid most stress on the mouse-virulence of the organism. From this he concludes that animal virulent organisms in the tonsils are a clear indication for tonsillectomy. PELZ.

Specht, of Kiel, in the Archiv. f. Ohrenh., Jan., 1929, 1-120, discusses the problem of cerebellar abscess without cerebellar symptoms. Neumann collected such cases and found 9.4 per cent without symptoms. Specht reports a case in which puncture and aspiration gave pus at 6 c.m. depth. After operation, cerebellar symptoms developed and patient was on the road to complete recovery when two months later he suddenly developed pain in the occipital region, nausea, vomiting, neck rigidity, followed by death.

GUGGENHEIM.

Asuerotherapy is discussed in the Journal A. M. A., Nov. 9, 1929. Dr. Asuero, of Madrid, has been curing many cases of chronic ailments, at his Shrine, by using a form of cauterization of the sphenopalatine ganglion. He first uses his magnetic personality to semihypnotize the patient and while the patient is still under this spell, he applies electrocautery to the region of the sphenopalatine ganglion. Clare Ousley, in the New York World, relates many miraculous cures by Dr. Asuero. MYERS.

Brunner, of Vienna, in the October, 1929, Mschr. f. Ohrenheilk., reports a case of Ochronosis of the Temporal Bone. This disease is a very rare one, only 35 cases having been found in the literature to date. It was first described by Virchow, in 1866. It is a peculiar disease which manifests itself in arthritic pains, yellow pigment in the skin and sclera and a highly colored urine. It is thought to be due to faulty melanim and trypsanin metabolism. Brunner examined the temporal bone of such a case and found yellow pigment embedded in the drum membrane and in the bone of the internal ear. He compares this bone with that of otosclerosis and osteoporosis. FINEBERG.

BOOK REVIEWS.

Pathologie der Oberen Luft- und Speisewege. By Dr. Felix Blumenfeld and Prof. Dr. Rudolf Jaffé. First edition. With 288 pages and 108 illustrations. Leipzig: Verlag Von Curt Kabitzsch. 1929. Price Rmks. 48.

Blumenfeld and Jaffé are assisted by A. W. Fischer, W. Pfeiffer and Berberich, Frankfurt a/M.; Schlossberger, Berlin; Siegmund, Cologne; Corten, Hamburg; Hellman, Wurtzburg; Kelemen, Budapest.

This is the first of two volumes and includes excellent clinical-pathological discussions on the role of constitution and heredity in diseases of the upper respiratory tract; changes caused by extreme factors; circulatory and degenerative diseases and developmental anomalies.

The outstanding features of this new book are the close co-operation between clinician and pathologist, the excellent colored reproductions, the complete bibliography, and the attention paid to some of the obscure diseases of the air passages.

Wound healing, inflammation and tumors will be dealt with in the second volume.

This work is slated to become a standard reference for otolaryngologists from both the clinical and pathological viewpoint. M. F.

Das Ohr und die Entstehung der Sprache und Schrift. By Pietro Tullio. Transl. by A. Jellinek. Berlin-Wien, 1929; Urban & Schwarzenberg. Pp. xi and 460.

The title of the book is misleading. Very little is said in it about the phenomena of hearing as suggested by the word "Ohr". The author restricts himself to a mere quoting of the common "harp theory" originated 150 years ago by Cotugno and nowadays usually, and falsely, attributed to Helmholtz: Helmholtz only adopted it after much vacillation. The theory of Bonnier and Meyer, which does not assume any kind of tension in the cochlea, but only hydraulic functioning, and which requires a little more mathematical thinking for its comprehension than the resonance theory of Cotugno and Helmholtz, the author leaves entirely unmentioned, like most biologists. The last 80 pages of the book give a summary of the present status of the phonetic and linguistic sciences. A more correct title of the book would have been "the possibility of stimulating the utriculus, the sacculus and the ampullae by long-continued sounds and causing thereby posture reactions". It is doubtful if such reflex actions deserve to be called "auditory".

The main part of the book is divided into two main parts. In the first the author gives a very good account of the work of several centuries on the possibilities of mechanical displacement resulting from acoustic attraction and repulsion. He reports some novel experiments of his own in this line. He then describes how mechanical disturbances with consequent stimulation of the sensory structures may result from sound waves, sufficiently prolonged, which act on the labyrinth. This may be called a perception of direction with a postural adjustment. In the second part, the author quotes very elaborately from the literature on reflex actions of mammals and birds and reports his own observations on the consequences of destroying various parts of the labyrinth. All of this makes quite interesting reading; but the author mixes up the discussion by generalizingly calling it all "sound localization", forgetting that localization is also possible for sounds of minimum duration, in which case reflexes apply entirely different from those in which this book is interested. In general, the author shows a tendency to exaggerate the importance of the particular reflexes in which he happens to be interested. He seems to think that these alone "determine the muscle tonus" and even "give the human mind the capacity of logical representation". The reviewer retorts that no particular sense organ should or can be regarded as the one on which exclusively the so-called "human mind" is founded. MAX F. MEYER.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

April 24, 1929.

(Continued from November issue, page 776.)

Bilateral Bronchial Fistula; Two Cases. Dr. J. D. Whitham.

Mrs. H., a nurse, age 38 years, was first seen June 15, 1924, when she complained of discharging fistulas on each side of her neck. She said that she had had this trouble all her life; that on the right side discharged continuously and caused much annoyance; that on the left discharged only occasionally.

A small, discharging opening was noted on each side along the anterior borders of the sternomastoids, about 10 c.ms. above the clavicles. A probe could be passed into each tract, but could not be made to enter the pharynx. The passage of the probe was attended by exquisite pain and spasms of coughing. The right tract was injected with barium paste; the tongue was then depressed and the barium was seen emerging from the tonsillar sinus at the lower pole of the tonsil. A Roentgenogram then taken showed the course of the tract very nicely.

Two weeks later, the tract was removed after injection with a solution of methylin blue. The dissection was easy until the bifurcation of the carotid artery was reached; here the tract was almost hair-like in thickness. Healing was complete.

In this case it would not have been practicable to follow the method advised by von Hachn, which resembles the Mayo operation for varicose veins of the leg, being carried on, in brief, as follows: After the carotid bifurcation is passed, a filiform bougie is passed through the fistula, and one end is brought out through the mouth. The other end is tied to the stump of the tract. By traction on the mouth end of the bougie, the tract is pulled out into the pharynx and cut off.

The other patient has a practically identical condition. The two cases are both bilateral branchial fistulas. The anatomical relations of this type of fistula are constant and their removal is not especially difficult. From the skin of the neck, often as low as the clavicle, it extends upwards to the border of the sternomastoid muscle; thence it passes to the hyoid region, where it crosses the digastric muscle, passes then to the bifurcation of the carotid artery, thence goes under the stylopharyngeus and styloglossus muscles, crossing the ninth and tenth cranial nerves, and enters the throat in the tonsillar region.

Tuberculosis of Tonsil and Pharynx. Dr. H. R. Leshin.

H. H., a man age 27 years, was admitted to the Manhattan Eye, Ear and Throat Clinic of Dr. Harmon Smith on Feb. 25, 1929, complaining of sore throat and pain, with some difficulty in swallowing, the trouble being of two weeks' duration. He had received private treatment for this sore throat, but with no improvement. He gave no history of previous attacks or other serious illness, with the exception of an injury to his right eye, resulting in its removal six months previously.

Examination showed the mucous membrane of the pharynx, the soft palate and mouth to be pale; the left tonsil presented a large, deeply excavated ulceration, with some exudate; this ulceration involved a good part of the posterior pillar. The epiglottis was edematous, red and infiltrated; the arytenoids also were infiltrated and congested.

Pathological Examination: Smears from the ulceration in the tonsil showed no Vincent's infection. The blood Wassermann and Kahn tests were negative.

March 2, 1929: Temperature, 100.2°; pulse, 120; respiration, 22. Mar. 4: Biopsy of the tonsil (left): The specimen consists of a small fragment of tonsil, which on microscopical examination showed numerous tubercles and multinucleated giant cells. March 2: X-ray of chest showed an acute miliary tuberculosis.

Diagnosis: Acute miliary pulmonary tuberculosis, with secondary involvement of the larynx and left tonsil.

DISCUSSION.

DR. IMPERATORI: One should be very careful, from a laryngological standpoint, to make a diagnosis of tuberculosis on the evidence of a tonsil that is suspicious. In those institutions, such as the Post-Graduate Hospital, where all tonsils are sectioned and examined microscopically, there is a large percentage that show tuberculosis with no evidence elsewhere in the body; so that if a laryngologist made a diagnosis on the findings of tubercle in the tonsil, he might be in error.

DR. M. C. MYERSON: I would ask the question as to whether the patient did not develop acute miliary tuberculosis following manipulation of the tonsil incidental to the medication? I have seen two cases after a tonsil with unsuspected tuberculosis had been operated upon. One can very readily make a diagnosis of tuberculosis of the tonsil on its clinical appearance. The tuberculous tonsil or palate has a distinctive pale yellowish-red color that does not shade into a darker periphery, but continues into the surrounding tissue with the same intensity of coloring. Whereas the color of Vincent's angina is darker and the periphery is much darker than the lesion itself. The paleness is lacking. In addition the tuberculous lesion is coarsely irregular and does not bleed as readily as a Vincent's lesion when manipulated. The Vincent's lesion has a finely granular base.

DR. R. C. HOWARD: About four years ago I showed a patient from Dr. MacKenty's clinic whose tonsil was practically extirpated by the process. Finally a section showed tuberculosis, and we also found pulmonary tuberculosis. We treated the man with transfusions and the quartz lamp locally to the ulcer and the tonsil lesion healed promptly, but several years later he died of pulmonary tuberculosis.

DR. M. J. MANDELBAUM: Last week a patient presented a peculiar ulcerated appearance of the tonsil, and I examined him for tuberculosis, Vincent's and lues, was negative for the last two, and three days later he died of acute tuberculous meningitis. He had no pulmonary symptoms.

DR. J. A. MILLER: Twenty years ago I turned over to Dr. Holden 1,000 tonsils and he said that 20 per cent of them were tuberculous.

DR. J. I. KLEPPER: In cases of Vincent's angina I always try to find some other cause; I think that Vincent's angina is secondary.

DR. LESHIN: I would like to assure Dr. Myerson that the X-ray of the chest taken two days before the biopsy proved it to be acute pulmonary miliary tuberculosis. Then, also, Dr. Ornstein said it takes six days to two weeks for tubercles to develop. So that the removal of a piece of tonsil tissue for pathological examination could not possibly have caused the pulmonary involvement, in view of the X-ray findings taken two days prior to the biopsy.

DR. G. G. ORNSTEIN: I was fortunate in seeing this case about a week after Dr. Leshin saw it, and was interested because a case of pulmonary tuberculosis was diagnosed first by a laryngologist after passing through the care of many physicians; despite the extensive involvement of both lungs, there were very scant signs; the patient complained of no cough and there was no expectoration. When I saw him he had a further extension of pathology in the form of miliary tubercles over the soft palate; the sputum was negative, which so frequently happens in miliary forms of tuberculosis. I have seen cases of this type before where the infection has started in the gastrointestinal tract, with miliary tuberculosis developing and involving the soft palate and larynx, X-ray examination at that time being negative. Where you have a miliary tuberculosis developing it sometimes takes six to eight weeks before the process can be visualized by the X-rays. Unless you are aware of the above, with the

X-rays negative, you do not suspect that you are dealing with a tuberculous process. A negative X-ray does not rule out acute miliary tuberculosis. I have seen the patient after he was transferred to Bellevue, where he has developed a tuberculous meningitis and is dying. Up to the present time he has a negative sputum.

During the past 10 years of work in tuberculosis I have been impressed with the fact that I have rarely seen tuberculosis of the larynx without pulmonary involvement. One of the most important points when there is a question of whether you are dealing with tuberculosis, malignancy or syphilis—long before the biopsy is taken, you should have an X-ray examination of the pulmonary fields. Except in the forms of tuberculosis which occur chiefly in infants or young adults, if you have evidence of tuberculosis in the lungs, the chances are that you are dealing with a tuberculous larynx.

Mucocele of Frontal Sinus. Dr. J. G. Strickler (by invitation).

Three years ago, P. B., white female, age 25 years presented herself at Dr. McCullagh's clinic, complaining of fairly severe frontal and some occipital headache on the left side; worse at night. Examination of the nose revealed nothing special. As routine, she was sent to the X-ray department. Report follows: The frontals are large and deep. The left shows a mild involvement from granulations or exudate, and there is a shadow in the left frontal sinus, which resembles a tumor. The right frontal is clear. Left ethmoid shows moderate involvement from granulation. Right ethmoid is clear. Left antrum shows a mild involvement. Right antrum is clear.

X-ray report, four days later: "There is an area, left frontal sinus, which is suspiciously like a mucocele. Measurement, 30x35x18 m.m."

X-ray, three weeks later: Lipiodol injected in the left frontal sinus passes around a circular mass corresponding to a shadow seen in the first examination showing that this mass occupies cavity of the frontal sinus, and is not partitioned in the sinus. About two weeks later, further X-ray showed the lipiodol still in the frontal sinus above the tumor mass. The blood Wassermann was 1-plus; the Kahn, negative.

Blood count negative: R.B.C., 4,260,000; white C., 8,100; polys., 69 per cent; small lymphs., 23 per cent; large lymphs., 8 per cent; hemoglobin, 73 per cent.

The frontal sinus was opened by the usual external frontal sinus incision, and a smooth, round mass corresponding in size and location to the X-ray picture was found. It ended in a ribbon-like, nonpatulous stork in the inferior portion, which ran down the nasofrontal duct to terminate, fan-shaped, in the region of the bulla. This was removed, and a communication established with the nose for drainage. The mass contained a thick, mucoid material.

Case 2: A little over two years ago, P. H., white male, age 52 years, presented himself at Dr. McCullagh's clinic complaining of continuous frontal headache and some occipital headache. The pain bore no relation to time of day or season. Nasal examination revealed nothing of special interest.

Routine X-ray examination brought the following report: The frontals are extremely large and deep. The left extends to outer border of the orbit, where there seems to be a large separate cell. The left also shows a moderate involvement from granulations, which do not seem to involve a separate cell. There are large supraorbital cells, which extend well back over the orbits. There is a shadow in the floor of the right frontal resembling a mucocele. Measurement, 18x18x12 m.m. The ethmoids show a moderate involvement, especially on the left, with some swelling of the periosteum. Both antra show a severe involvement. The sphenoids are small and clear.

Later an attempt was made to introduce lipicdol into the frontal sinus. Fluoroscopic examination showed the cannula in the proper position to enter the sinus, but on obstruction was encountered, preventing entry. The lipiodol did not enter the frontal sinus. The same operative procedure was carried out as for Case 1. A mass corresponding in size and location to the X-ray picture was found. The Wassermann in this case was also negative.

Points of interest: 1. In both cases ,the diagnosis was made by the X-ray. 2. Both patients complained of some occipital headache. 3. One of the patients

had an open ostium, the other a closed ostium to the frontal sinus. 4. There was no history of trauma in either of these cases.

DISCUSSION.

DR. A. LOBELL: I was fortunate to see the operation Dr. Strickler performed, and I would like to say that he should be congratulated on his successful operation, management, result and excellent presentation. It is needless to mention that Dr. Law equally deserves a great deal of credit for the prompt detection of the mucoceles on the X-ray films.

Some time ago I reported a mucocele of the antrum and discussed the relationship between mucoceles and cysts. The textbooks give us a conception of a mucocele which differs from reality. For instance, if anyone were to ask, "What is a mucocele?" the answer would probably be that after the occlusion of the ostium a gelatinous material accumulates within the sinus, and as a result of the gradual intrasinus pressure the walls of the sinus become very thin, soft and yield. A protrusion of the sinus wall ensues, and the patient seeks relief from the grotesque disfigurement. This conception is simply perpetuated by carrying over the old pictures in the textbooks. That was the situation in the pre-X-ray days, when there was no opportunity to recognize a mucocele before it made itself evident by a protrusion.

However, the X-ray has enabled us to diagnose the mucocele in its early stage of development, and we discovered that an occlusion of the ostium is not the direct and primary cause of the mucocele. The real origin of the mucocele is due to a cystic formation of the mucosa in the ethmoid or in the floor of the frontal sinus which gradually occludes the ostium during growth and expansion within the frontal sinus. If occlusion of the ostium were the primary cause of a mucocele, this condition would occur more often because opportunities for occlusion are numerous. The fact that cystoceles are more frequent in the frontal than in the maxillary sinuses speaks for cystic degeneration. A floor of a sinus is subjected to irritation by products of inflammation as a result of gravity and stagnation. This situation makes the floor a most favorable soil for cystic degeneration. Inasmuch as the ostium happens to be located in the floor of the frontal, it is only natural to expect that the ostium would become occluded very soon, whereas the reverse is true of the cyst rising from the floor of the antrum. Here the mucocele can grow to its full capacity before it has a chance to reach the ostium, and occlusion, if at all occurs, would be in the late stage. Hence, the diagnosis of maxillary cysts were always made before occlusion of the ostium took place. From this reasoning, we may conclude that essentially there is no real pathologic distinction between a cyst and a mucocele. The former term designates the condition before the ostium becomes occluded and the latter after occlusion takes place.

JAN 7 1930

INDEX NUMBER

Vol. XXXIX DECEMBER, 1929 No. 12

THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
DEVOTED TO DISEASES OF THE

EAR - NOSE - THROAT

[redacted]

FOUNDED IN 1890 BY

DR. M. A. GOLDSTEIN

Managing Editor and Publisher.

DR. MAXWELL FINEBERG, St. Louis,
Assistant Editor.

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For Contents See Page 1.

Subscription, $6.00 per Annum, in Advance.

Foreign Subscription, 35 Shillings per Annum, Post Free.
Single Copies, 75 cents.

PUBLISHED BY THE LARYNGOSCOPE CO.

3858 Westminster Place, St. Louis, Mo., U. S. A.

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